

**SICK CHILDREN: DIAGNOSIS  
AND TREATMENT**

# SICK CHILDREN: DIAGNOSIS AND TREATMENT

*A MANUAL FOR STUDENTS  
AND PRACTITIONERS*

By

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THIS BOOK IS PRODUCED IN COMPLETE CONFORMITY  
WITH THE AUTHORIZED ECONOMY STANDARD.

## PREFACE TO THE FIFTH EDITION

Despite the war years much progress has been made in paediatrics. This has necessitated considerable revision of a number of sections of this book.

The chapter on the Sulpha drugs has been thoroughly revised and a new portion added on the use of penicillin.

Although no special section has been devoted to child psychology references to the management of the child and advice to the mother are made throughout and the importance of the psychological aspect of many problems is stressed.

The most recent views on hæmorrhagic disease of the new born and erythroblastosis foetalis have been included together with the latest knowledge on the vitamin requirements of infants and children.

Newer views on the treatment of colic disease are also mentioned. The section on gastro-enteritis has been thoroughly revised and rewritten as has also the section on epidemic jaundice. The chapters on diseases of the circulatory system and infectious diseases have been largely rewritten and, throughout the book, care has been taken to include the most up-to-date references.

The author is much indebted to the Registrar General's Department for up-to-date statistics, which have been included throughout and also to Dr W. W. Payne for providing details of normal laboratory data.

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## PREFACE TO THE FIRST EDITION

In this Manual I have attempted to deal with the commoner diseases of childhood as completely as the limits of space allowed keeping constantly in view the needs of the senior student and the general practitioner. The reader I hope will find that the descriptions of symptoms though concise are adequate nor have ætiology and pathology been neglected but, as the title suggests the main stress has been laid on diagnosis and treatment as the surest way of making the book practical and helpful.

I have to thank Drs. Still, Cameron and Poynton for the loan of illustrations and for kindly help and Dr. Robert Hutchison for the guidance I have found in his *Lectures on Diseases of Children* and *Food and the Principles of Dietetics* both published by Messrs. Edward Arnold & Co. to whom I am indebted for permission to quote two tables from Garrod, Batten, Thursfield and Paterson's *Diseases of Children*. I must also thank the Editors of the *British Medical Journal*, the *Lancet*, the *Post Graduate Medical Journal*, the *Archives of Diseases in Childhood* and the *Practitioner* for permission to reproduce illustrations and papers of mine that have appeared in the pages of those publications. Nor must I fail to acknowledge the courtesy of Messrs. Constable & Co., Ltd. in allowing me to copy some of the food tables and diet sheets that were compiled for Paterson and Forest Smith's *Modern Methods of Feeding in Infancy and Childhood* which deals with the subject of infant and child feeding much more at length than is requisite in a book on Sick Children.

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D.P.

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# SICK CHILDREN: DIAGNOSIS AND TREATMENT

## CHAPTER I

### NORMAL AND ABNORMAL GROWTH AND DEVELOPMENT

#### EXAMINATION OF A NORMAL CHILD

Much greater care is necessary in examining a child than an adult. The physician is helped by the adult's own description of his symptoms, but with a child he must very often depend on the observations of the mother or nurse, which may be quite helpful or exactly the reverse.

For a thorough examination an infant or child should be quiet and happy. The co-operation of the mother or nurse is necessary to keep it amused. Infants and children from the age of 6 months on are happiest when sitting up on the mother's knee and after the age of 1 year as a rule they tend to cry if forced on to their backs. Throats should be examined last of all, since all children are apt to object. Percussion and auscultation of the chest should be performed with warmed fingers, and a warmed stethoscope nose piece. Very often a play of examining the mother's chest first, before attempting the child's, will reassure it. Above all there must be no hurry or brusqueness if the examination is to be made without tears. The physician will be amply repaid for the expenditure of a few moments at the beginning, spent chatting with the parents and the patient so that the child becomes accustomed to the physician and the voice and gains confidence. A consoling room with a few toys and objects of interest to children of various ages will remove much of the terror of a visit to the doctor. The author assures all young practitioners that they should not be discouraged, despite early failures, for practice is the only real road to success.

No examination is complete which does not include a careful inspection of the throat and ears, even if a struggle is involved. Finally, it is essential to examine the urine and, where possible, to see a motion recently passed.

**The head.**—Note the shape of the head, and remember that at birth the circumference of the head should be 13 to 14 inches, at 6 months 17 inches, at 12 months 18 inches, at 18 months 18½ inches, at 2 years 18¾ inches, at 3 years 19¼ inches, at 4 years 19½ inches, at 5 years 20½ inches, at 10 years 21 inches, and at 16 years 21¾ inches. A very small head is important as it suggests microcephaly or failure of the brain to develop. A head which is definitely too large suggests some form of hydrocephalus.

The shape of the head is also important. In rickets it is bossed and of the

so called 'hot cross bun' shape or large and square in hydrocephalus the forehead is overhanging and after certain forms of birth injury, or with the head moulding in a difficult labour there is asymmetry the whole being skewed to one side or the other. One side of the head is larger than the other in hemiatrophy.

**The fontanelle**—The fontanelle should close about the 18th month but occasionally it closes or almost closes earlier. Very early closure, especially in premature infants suggests microcephaly. It may be widely open in hydrocephalus severe rickets or osteogenesis imperfecta the child often reaching the age of 2 or 3 years before it closes.

**The hair**—Normally the infant has a little soft down at birth, but occasionally there is a thick black brush or wig. By 6 months there is a good growth of new hair. In rickets and still more in cretinism, the hair becomes straight and dry. In the mongol the hair is straight but softer. The scalp should always be examined to exclude ringworm and patches of alopecia, scurf and impetigo. In newborn babies a swelling of the scalp often appears at the site of presentation this is the *caput succedaneum* and is really an oedema of the subcutaneous tissues (see p. 59). Occasionally a cephalocele may form (see p. 59).

**The eyes**—The shape of the eyes is important. Apart from such abnormalities as one blue eye and one brown one congenital malformations should be sought such as reflection lack of the light from an opaque surface suggesting cataract. In mongolism there is a tendency to squint with an oval or slanting shape to the eyes and an exaggeration of the inner canthus. Other causes of squint are encephalitis lethargica meningitis and brain tumours. Marked external squint may be present at birth or may be acquired as the result of some error of accommodation. Slight concomitant squint is the commonest type in childhood, and may be of little or no importance. It usually passes off as a child gets older. There is a tendency to squint in every new born infant this is of no significance and is quite transient. Short lateral or vertical nystagmoid movements should be looked for as these are significant in the diagnosis of cerebral lesions and are occasionally found accompanying rickets and spasmodic nutans<sup>1</sup> (head nodding) in infants.

The normal infant should begin to follow a light at 3 or 4 weeks of age, and should see objects clearly by the time he is 2 months old. If he is not attracted by or cannot focus on a light after this age he is likely to be blind from a local eye blindness or from cerebral agenesis or degeneration of the cerebral cortex. *Piosis* may be congenital, in which case it is often familial or acquired due to some defect in the nerve supply to the eyelid, or the eye may be partially closed from oedema, due to some inflammatory condition.

**The nose**—The shape of the nose is important. In congenital syphilis with a chronic nasal discharge appearing shortly after birth the bridge rapidly becomes infected and is destroyed and a permanent depression results. The pinched in nose of 'adenoids' or nasal obstruction, often accompanies chronic sinusitis, and is easily detected, the

under sized ill developed nose of the average asthmatic child is striking. In pneumonia the alae nasi can be seen to work with each inspiration.

**The ears**—Care should be taken to ascertain that the child can hear. If there is any doubt about his mentality simple tests should be made to see whether he hears sounds and how well. Find out whether coming into his room at night and making a noise wakes him, clap your hands loudly or drop something behind him, speak to him or whistle to see whether he will turn towards these sounds. An examination of the drums should be made in every case as a routine especially in cases where there is a fever without apparent cause. Wax may obscure the examination and must be removed with hydrogen peroxide and water equal parts put in several times daily, drying thoroughly with cotton wool or by syringing with a solution of sodium bicarbonate one teaspoonful to a glass of tepid water.

**Complexion and face**—The normal child is pictured as pink checked and fair skinned but this varies with his parentage. The new born infant is red faced then within a week or two his complexion becomes very pale and remains so until he is 2 or 3 months old at least when a little colour begins to appear. Some children are always pale faced although not anæmic and especially children with auburn hair. Dark haired families often have very white skins.

In rickets tapping the face produces a mild spasm of the muscle (Chrostek's sign). Extreme thinness of the face is a sign in lipodystrophia progressiva. In the extreme emaciation of the marasmic infant the sucking pads protrude in the cheeks the rest of the face being thin and pinched.

**The mouth, lips and gums**—The lips are cracked and excoriated in congenital syphilis (rhagades) and dry and cracked in febrile conditions of all sorts. Herpes may appear as small blisters or crusts. The mouth is carp shaped, or turns down at the corners in mongolism and is open widely, allowing the huge tongue to protrude, in cretinism. The lips may be pale and almost colourless in blood diseases or dark red or cyanosed in heart disease, asthma or pneumonia.

In scurvy the gums are swollen red and hæmorrhagic. In ulcerative stomatitis they may almost cover the teeth. Purpuric patches or hæmorrhages into the gums may be seen.

**The throat**—In examination of a child's mouth and throat it is best to wrap him in a blanket or shawl or else he should sit on his mother's knee. She should take both his hands in one of hers and hold his head firmly against her by placing her other hand over his forehead. In this way the tongue can be depressed with a teaspoon, and the mouth and throat can be inspected in comfort using a pocket torch. The number and condition of the teeth should be noted and a glance should be sufficient to detect ulcers. Depressing the tongue brings the tonsils into view and the presence or absence of membranes or of tonsillitis can be noted.

**The glands**—By palpating at the angle of the jaw the enlarged tonsillar glands can be felt, and the glands draining the adenoid region can be detected behind the sternomastoid. The glands in the axilla and inguinal region should be palpated as a routine, and a careful examination made of the

abdomen especially in the region of the mesentery, for tuberculous adenitis. Enlarged glands in this region suggest blood diseases such as leukaemia and Hodgkin's disease or tuberculosis, syphilis or a new growth. Inflammatory conditions in the fingers or toes would of course give rise to adenitis in the axilla or groin.

**The neck.**—Palpation of the sternomastoid will often reveal a small hard lump due to a hematoma into the sternomastoid at birth. Generally this disappears completely within the first year. A tendency to wry neck may follow or there may be a congenital shortening of the sternomastoid muscle causing the head to be held to one side. In such cases the child should be so placed on its pillow as to press the head into the correct position in this way operation is often avoided. In cerebellar tumour the occiput is tilted towards the side of the lesion. Head retraction is seen in cases of meningitis and other cerebral irritation, or there may be a stiffness of the neck only, as in poliomyelitis, spinal cancer and inflamed neck glands.

**The chest and its shape.**—The chest should be examined first by palpation the ribs being felt for the presence or absence of beading. In rickets beading is most marked at the costochondral junctions. Backward dislocation of the sternum suggests scurvy. In osteogenesis imperfecta there is a marked tendency to fracture of the ribs. If the chest be examined posteriorly the shape of the spine can be seen. Kyphosis suggests rickets if reducible or spinal cancer if irreducible. Scoliosis suggests congenital malformation of the vertebrae, or may be the result of poliomyelitis or rickets. It is most commonly due however, to faulty posture and general hypotonia of the muscles. Pigeon chest results from rickets, together with some form of respiratory obstruction or disability, such as adenoids or broncho pneumonia. In asthma the chest may be barrel shaped. Marked depression of the sternum is seen in thin, hypotonic children who have had rickets and whose chests are sucked in along the line of the diaphragm (Harrison's sulcus). Depression of the ribs or bulging of the chest on one side or the other suggests old rickets.

**The lungs.**—The breath sounds in children are harsh and expiration can be heard to some extent as well as inspiration. In the teething child who is salivating sounds referred from the trachea are heard throughout the chest. In many cases a slight diminution of air entry or an occasional crepitation may indicate considerable underlying disease. On the whole slight changes are much less significant than they are in the adult. Percussion should be light and not carried to any extent below the angle of the scapula as the liver and kidneys may give rise to dullness which will mislead.

**The heart.**—Percussion of the heart is difficult and not very satisfactory. Gross abnormalities in size can be detected by palpating the apex beat and feeling for the general cardiac impulse to the præcordia. On auscultation both the aortic and pulmonary sounds are best heard to the left of the sternum. The presence or absence of a thrill over the præcordia should be noted in every case, if present, it suggests congenital morbus cordis.

**The abdomen.**—In the infant the abdomen is normally full and thus

fullness persists sometimes into the second year, but by the age of two the abdomen should be flattened and no longer pendulous. An unduly large abdomen may be due to a full bladder, rickets, constipation, celiac disease, tuberculosis, starch indigestion, renal or other tumours, or gross enlargement of the liver or spleen. Wind swallowing and Hirschsprung's disease should also be considered. The condition of the navel should always be inquired into, especially in the new born. Umbilical sepsis during the first week is the cause of a long chain of symptoms, from jaundice to meningitis, pneumonia and general septicæmia.

On palpating the abdomen the liver should be outlined. Normally this extends one to two fingers' breadth below the costal margin. In celiac disease it frequently cannot be palpated. In cirrhosis it can be felt firm and hard, and enlarged to a greater or lesser extent. Great enlargement of the liver is found in tuberculosis, infective hepatitis, anemias, leukæmia, syphilis, von Gierke's disease, neoplasms, and diabetic hepatosis. Subdiaphragmatic effusion or growths may also displace the organ downwards. The spleen is normally not palpable. When it is, it suggests congenital syphilis, tuberculosis, infective hepatitis, malaria, new growth or blood diseases, but any passing acute infection may give rise to some transitory enlargement. Deep palpation of the abdomen for enlarged kidneys or other masses should be carried out. The child should be examined in a good light, standing, if possible, to allow herniæ to be seen, and the presence or absence of the testicles in the scrotum should be ascertained by palpation. Mucositis should be looked for, and pin point meatus or meatal ulcer in circumcised children.

**The legs and arms.**—The shape of the legs and arms should be noted. Knock knee or bow leg suggests rickets. Abnormal shortness of the arms and legs is present in achondroplasia. Walking with a *scaddling gait* suggests achondroplasia or congenital dislocation of the hip on one or both sides, or *pseudo hypertrophic muscular dystrophy*. *Limping* may be seen in simple trauma of the foot, leg or hip, or in tuberculous disease. Pain on movement of the limbs in infants suggests scurvy. A *rolling, staggering gait* suggests cerebellar tumour or post diphtheritic paralysis.

**The knee-jerks.**—An examination of the knee-jerks is of the greatest importance. In cerebral lesions, such as spastic diplegia, the knee jerks are much increased and there is a spasm or spasticity of the lower limbs. In chorea the knee-jerk is prolonged or sustained. Absence of the knee jerks is seen in poliomyelitis, Friedreich's ataxia and post diphtheritic paralysis. Babinski's reflex is of less importance in infants, it tends to be extensor normally. Ataxia of the arms is shown in the nose finger test, both in cerebellar and cerebral lesions.

#### NORMAL DEVELOPMENT AND TRAINING

**Heredity.**—In the year 1865 the Abbé Gregor Mendel studied the effect of crossing different varieties of peas, and formulated what is called "Mendel's Law of Heredity." He noted that some characteristics were "dominant" and some "recessive," and according as they were dominant or recessive so there was a greater or lesser tendency for them to be transmitted. Roughly, the mating of two dominants produces dominants, and the mating



of two recessives perpetuates the recessive characteristics, but the mating of a dominant and a recessive will produce 25 per cent pure dominants and 25 per cent pure recessives whereas the remaining 50 per cent have both characteristics

Inherited abnormalities of man and the rare normal characteristics fall into three main groups, according to Cockayne<sup>1</sup>. The first consists of those which are transmitted direct from parent to child for generations and are often called hereditary. The second are those in which several members of one family may be affected in which case they are known as familial. In the third group the tendency is sex linked that is it may pass to the males through the female children, as in hæmophilia. Some of the conditions which Cockayne considers hereditary are xeroderma pigmentosa von Recklinghausen's disease tylosis palmarum et plantarum and brachyphalangy albinism deaf mutism bifid thumbs and coloboma of the iris arachnodactyly and dislocation of the lens polydactyly, obesity and cerebro-inacular degeneration forming the triad known as the Lawrence-Moon Biedl syndrome pseudo hypertrophic muscular dystrophy hæmophilia red green blindness ichthyosis simplex hare lip and cleft palate and cystinuria. He thinks that an environmental factor operating during embryonic life makes manifest the genetic factor to produce Mongolian idiocy spina bifida and congenital pyloric stenosis.

Some of these abnormalities are extremely rare and when consins marry each having the tendency to them the proportion of their children showing the abnormality is exceedingly high. It is estimated that the proportion of first cousin marriages in a community is between one half of 1 per cent and 1 per cent. It is not to be wondered at therefore that these rare abnormalities persist.

Immunity to disease is probably largely an inherited characteristic, and by a process of natural selection the non immunes are reduced.

The work of the paediatrician brings him into close contact with parents and he very soon sees in their children many of the characteristics of both. Adiposity is often inherited and allergic tendencies hay fever and asthma can be seen running in a family. A tendency to a weakness of one organ such as stomach or kidney or mental acuity an even disposition good looks and many other characteristics are inherited from both sides of the family. It may well be asked what part environment plays. There is no doubt that good feeding and hygiene together with proper management provide the opportunity of making the most of a child's talents. In the author's experience heredity is infinitely more important than environment in the final result.

Adoption of children—Since the adoption of children has become legalized it is now practised on a large scale. The greatest care should be taken to exclude children who may have inherited undesirable tendencies. The family history should be carefully enquired into for asthma and hay fever epilepsy, and mental trouble and a Wasserman test should be done to exclude syphilis. The mentality and appearance of the parents should be ascertained as almost all their chief characteristics may later be reflected

<sup>1</sup> E. A. Cockayne. Hereby in Carol Ratten and Thurnfield's "Diseases of Children" Arnold 4th ed. 1915 p. 1.

in the child. Fortunately, most of the definitely hereditary diseases such as hæmophilia and pseudo hypertrophic muscular dystrophy are extremely rare.

### HEIGHT AND WEIGHT

**Growth**—The normal growth of childhood is in three separate phases or cycles each composed of a period of gradual acceleration, peak and retardation. The first is to be observed in the 1st year of post natal life (babyhood), the second with its peak at the 6th or 7th year (second dentition) and the third is the adolescent spurt (puberty).<sup>1</sup>

On the whole girls grow more steadily than boys. The limbs of the infantile skeleton are short in comparison with the trunk. After the age of 7 years the lower limbs are proportionately longer than the trunk. This is accounted for by the slow growth of the cartilage of the vertebrae compared with the cartilage of the limbs that is at the knee and hip, elbow and shoulder. No longitudinal growth takes place in the body of the bone but only at the epiphyses.

**Factors influencing growth**—Undoubtedly heredity is of great importance. The most important factor however is the action of the anterior lobe of the pituitary gland and its growth hormone. It seems to have a stimulating effect on the growth of cartilage as on the other portions of the body. An absence of thyroid secretion prevents normal growth and an excess of thymus gland stimulates it. All four vitamins A, B, C and D, influence growth in their own way. Vitamin A affects epithelial structures, vitamin B the nervous tissue, vitamin C the vascular system and vitamin D cartilage and its calcification. Diet is important particularly in relation to the biological value of protein. Animal protein particularly in the form of whole milk, cheese, eggs, fish and meat is necessary to provide the essential amino acids.

**Factors limiting growth**—With the advent of sex maturity the epiphyseal junctions close and longitudinal growth ceases. The essential feature appears to be a maturation and senescent change in the growth cartilage, proliferation ceasing while the epiphyseal discs ossify.

Other factors of great importance in limiting growth are infective and metabolic diseases. Harris<sup>2</sup> finds that growth cartilages are inordinately sensitive to illness and that during the period of growth any serious interference with nutrition from starvation, acute infection or metabolic disease may induce premature senescence. The fact is registered in the structure of the bone by a transverse line of arrested growth. This line is heavily calcified and can be seen by X ray as a scar. A series of severe infections particularly the exanthemata in quick succession may result in dwarfism.

**Growth in height**—The normal child at birth is from 18 to 22 inches in length and by one year he is 28 to 30 inches long. From this time on there is a growth of roughly 2½ inches per year. Up to the age of 12 the girls are slightly shorter than the boys; at 12 and 13 they are slightly taller and from 14 years on the boy once more leads in height.

<sup>1</sup> H. Gardner Hill. Abnormalities of Growth and Development. (The Clinical and Pathological Aspects). *Brit Med Jour.*, June 1937, 1, 341.  
<sup>2</sup> *Archives Internal Medicine*, 1933, xxxviii, 34.

Growth in height, like growth in weight, is not steady, but goes ahead by leaps and bounds, nor is it definitely seasonal. Being confined to bed does not always cause more rapid growth. The height of normal children of various ages is shown in Table I.

TABLE I

HEIGHT OF A NORMAL CHILD FROM BIRTH TO FIFTEEN YEARS

Age	Boys Height (inches)	Girls Height (inches)
Birth	21	21
6 months	25	25
1 year	29	29
2 years	32	32
3	35	35
4	38	38
5	42	41
6	44	43
7	46	46
8	48	48
9	50	50
10	52	52
11	54	54
12	56	57
13	58	59
14	61	60
15	64	62

Various factors operate in retarding a child's growth and producing infantilism, dwarfism or stunting. On the other hand, gigantism is also seen in childhood.

**Weight of the normal child.**—An average infant weighs at birth about 7 lb. There is a loss during the first four to seven days of anything up to half a pound, but this is quickly made up, and by the tenth to twelfth day the birth weight is regained. Thereafter from 6 to 8 ounces a week are gained for the first five months, and about 4 ounces per week from five months to one year. At five months the infant has doubled its weight and at one year it has tripled it. In private practice much greater gains than these are found. It is not in the least uncommon for a 7 lb infant to weigh 18 lb at 6 months and 25 lb at one year. This does not necessarily mean that the child is more healthy, but very often it has thrived well because of the extremely good care bestowed on it.

The *regular weighing* of infants ought to be insisted upon, as it is only by weighing that progress can be gauged. For the first six months the infant ought to be weighed weekly, from six months to one year fortnightly, from one to two years monthly, and from thence up to 12 years every two or three months. No well cared for healthy infant should lose weight. The weight may tend to increase more rapidly one week than another, but there should always be a gain. Cutting teeth, a change of nurse or human environment or vaccination may all cause stationary weight temporarily.

In *older children* emaciation or wasting may be due to too great activity and to nervous exhaustion to dental caries with chronic indigestion, rheumatism, tuberculosis diabetes. The constitutionally over active child is extremely difficult to fatten and is a problem in practice. (See *Nervous Exhaustion*, p 265) Older children gain weight at irregular rates.<sup>1</sup> Some children gain in the summer only and are stationary or gain very little in the winter, and vice versa. The weight of normal children at various ages is shown in Table II.

TABLE II  
WEIGHT OF A NORMAL CHILD FROM BIRTH TO FIFTEEN YEARS

Age	BOYS Weight in lb	GIRLS Weight in lb
Birth	4	7
6 months	16	15½
1 year	21	20
2 years	26	26
3	31	30
4	37	34
5	41	40
6	45	44
7	50	48
8	55	53
9	60	58
10	67	64
11	72	71
12	80	79
13	88	87
14	97	101
15	111	103

### SIGHT AND SPEECH

Shortly after birth the child should follow a bright light with his eyes. Certainly by one month a normal child will follow a bright object, and will recognize his mother or nurse between the ages of 2 and 3 months. Objects placed in the hand can be grasped tightly from birth onward but the child is not able to reach out and grasp before 3 to 4 months of age. Things are held properly in the hands and passed from one hand to the other at the age of 6 to 9 months, and a normal infant of 15 to 18 months learns to feed himself with a spoon.

Speech is acquired slowly. By one year most infants are able to say *Ta* for *Thank you*, *Mama* and *Dada* and can imitate the sounds of various animals. Single words are clearly spoken by 15 months and short two or three word sentences by 2 years, when the child has acquired quite a large vocabulary. Variations in the rate of learning to speak are very great, some children articulating clearly at a year and forming long sentences by the end of the second year, while others are much more backward. Such variations in speech do not in any way signify mental abnormality. Small nursery rhymes can be learnt by 2½ to 3 years and the child is able to say his letters by 4 or 5 years. By the age of 5 to 6 years he can learn to write.

<sup>1</sup> Emmett Holt, *Observations on the Health and Growth of Children in an Institution*, *Amer Journ Dis Child*, July 1923, 1, 1.

**Walking**—The average child should sit up by 6 months, pull himself up in his cot into an upright position by 9 or 10 months and walk at 12 or 14 months. He should walk quite well by 18 to 20 months. Over fat children acquire these capacities much more slowly. This may be due only to their weight or in part to flabbiness or even to rickets. There is a feeling among the public especially among old fashioned nannies that a child should not be allowed to sit up stand or walk even when he shows a marked inclination to do thus himself. Experience is directly contrary to this belief and nothing but good can come from early progress.

### NORMAL DENTITION

**First set of teeth or primary dentition**—The date of the eruption of the different teeth varies but on the whole they are cut in the following order and about the following times:

- 1 Lower central incisors 5 to 10 months
- 2 Upper central and lateral incisors 8 to 12 months
- 3 Lower lateral incisors and lower and upper first molars 12 to 14 months
- 4 Lower and upper canines 16 to 22 months
- 5 Lower and upper second molars 24 to 30 months

**Delayed dentition**—As a rule, the teeth appear in pairs. Many abnormalities in appearance and type of teeth cut appear to be hereditary, and there can be no doubt that good teeth run in families.

Probably one of the chief factors in causing delayed and imperfect dentition is *rickets*. The disease is due to a lack of vitamins, fresh air, sunshine and proper food all of which contribute to cause defective calcification of the bones, including the teeth. This both delays the date of eruption and affects the structure. Suitable doses of cod liver oil a well balanced diet and plenty of fresh air and sunshine will guarantee as far as possible normal times of eruption and normal teeth.

**Symptoms of dentition**—Two extreme theories of dentition are advanced (1) that there are myriads of diseases and upsets due to cutting the teeth (2) that teething is responsible for nothing but the cutting of teeth.

Those who are fortunate enough to have infants under their charge in a hospital ward during dentition when the temperature pulse and weight are closely observed are able to form an excellent opinion. It seems that, varying with the individual this physiological process may become pathological, just as may childbirth.

**Fever**—Some children are inclined to be fretful and out of sorts, and actually have a temperature immediately before cutting a tooth without any other ascertainable cause of fever.

**Vomiting**—During the eruption of the first set of teeth especially between the ages of one year and 16 months when an attempt is being made to introduce more solid food there is often a strong tendency to vomit any solid diet. This tendency rights itself rapidly after the eruption of each tooth and is probably only mechanical because of the tender gums the child refuses to masticate the solid particles and swallows them whole.

**Diarrhoea**—When diarrhoea occurs during dentition the stools are characteristic they are loose, but of a good colour, and do not contain

undigested particles. In addition the infant does not fail to gain weight. Dietetic measures to check the diarrhoea such as reducing the feed or changing to some other food do not check the frequency.

*Neuralgia* —It seems reasonable to believe that dentition is accompanied by neuralgic pains. The face may become flushed on one or both sides one side being warmer than the other. Very often the infant bangs its head and apparently gets some relief. The hand is held to the side of the face and the sleep is restless and disturbed.

*Cough* —Children may get bronchitis at the same time as dentition but the common cough accompanying dentition is not bronchitic in the majority of cases. The saliva which normally flows from the mouth during the day flows to the back of the pharynx at night where it tends to occlude the larynx. Thus the child coughs at intervals especially during the night hours when placed on the back.

*Convulsions* —In a normal child convulsions never accompany dentition but they may occur in a rachitic or mentally defective infant.

*Face and skin eruptions* —Infants liable to eczema have acute exacerbations accompanying each tooth the eczema clearing up entirely when dentition is complete. Lichen urticatus and various other urticarial rashes also appear and disappear at this time.

*Treatment of the child while teething* —Since rachitic infants have more difficulty in cutting their teeth than non rachitic children their diet must be well balanced and the anti rachitic factors must be present including cod liver oil in some form. A routine purge for a few days will be found most useful especially glyster powder given at bedtime with a little milk of magnesia first thing the following morning. One of the liquid paraffin preparations may also be useful. If the disturbance at night is severe causing much loss of sleep chloral hydrate is recommended. (See Appendix p 397)

Great care must be taken before making a diagnosis of teething that all organic disease has been excluded. Pink disease is most often put down to teething. (See p 247)

### Secondary dentition (permanent teeth) —

First molars appear between the ages of	5	and	7
Central incisors	6½		8
Lateral incisors	7		9
First bicusps	9		11
Second bicusps	10		12
Cusps	11		14
Second molars	11		13
Third molars	16		17 or later

### TRAINING THE CHILD IN GOOD HABITS OF MICTURITION AND DEFECATION

The infant should be placed on his chamber at regular intervals after each feed almost from birth and an association should be firmly established in his mind between the feel of the chamber together with the position and the act of micturition or defecation. Gradually this association will become so fixed in his mind that the bladder and bowel are only emptied when he is placed on his chamber. As the feeds are spaced farther and

farther apart and become less and less fluid, control becomes more perfect. Even although the infant may pass his water between feeds, he should nevertheless be held out at the set times, until the bladder is again emptied. Much time should be devoted to this in the early months. A quiet insistence will greatly repay the mother or nurse.

Except for occasional lapses the normal child may be kept dry during the day between the ages of 9 and 12 months, and he should certainly be dry at night between a year and 18 months. Variations in the age when the bladder is controlled are however, common. Exacerbations of wetting occur during dentition or in extremely cold weather. Five minims of tincture of belladonna given three times daily at such times will aid the child in acquiring and keeping control. By 2½ years every normal child should be able to ask to be attended to during the day, and this should be on waking, after breakfast at dinner, at tea and at bedtime only. During the night some children require to be lifted up at 10 p.m., but this should be discontinued as soon as possible. Giving the last meal at 5 p.m. and no drinks afterwards is a very great help in getting such children to remain dry during the night.

**Defecation**—The child can usually be trained to pass his motions into the chamber at stated times at the age of 2 to 3 months. If he is constipated paraffin emulsion or some mild aperient may be necessary, but in the ordinary way a little gentle stimulation of the anus with a well vaselined finger tip or the tip of a soft rubber catheter will produce defecation. At 9 or 10 months the child should be able to sit and balance on his chamber and should be left there after breakfast and after tea for the purpose of emptying the bowel.

### PROPER CLOTHING

The clothing of the infant and child naturally varies with the climate and the customs of the people. Among the well to do, where houses are properly heated, less indoor clothing and more outdoor clothing is required than among the poor who need to wear more indoor clothing. Undoubtedly the children of the hospital class are as a rule enclosed in too many garments of an inferior quality, seven or eight layers of shoddy cotton being of less value than one or two woollen garments.

The infant requires a long sleeved, woollen undervest and a long flannel petticoat which can be folded over the feet and legs. Socks should be worn. A cotton or linen gown is usually worn outside and, during the winter months, a woollen jacket as well. Shortening should depend on the progress the child is making and his weight, not on his age. A large infant of 10 lb. may be safely shortened into woollen knickers and jersey when one or two months old, while in small premature infants this cannot be done until they are about 5 to 6 months old. It is a great advantage to the infant to be free to kick and develop his limbs, and when in doubt it is always best to do away with the encumbrance of the long clothes. The infant's binder should be discontinued at the end of a fortnight, a knitted abdominal belt being substituted, and this in its turn discontinued after one to two months. Too often the binder "works up" to the child's thorax, compressing it and being of no practical value.

Between the ages of one and two years summer or winter weight combinations or woollen vest and pants should be worn next the skin ( "Chilprufe" is a good make ) Woollen knickers and jersey during the winter and cotton suits during the summer are most suitable In very warm weather silk or cotton combinations may take the place of the woollen ones, but in climates where the temperature drops towards evening the greatest care must be taken in shunning wool

**Caps and bonnets.**—The infant born in the summer need not wear a bonnet or cap when taken out but those born during the winter require some protection until the hair has grown sufficiently All children require woollen caps or some form of head covering during severe weather

**Shoes.**—At the age of one year when the infant begins to walk, it is best to provide him with shoes having firm, non-slipping soles and small heels A good make is the "Start-Rite" shoe, but there are various other makes which are equally satisfactory The heels should be narrow enough to fit snugly, whereas the toe should be broad, allowing plenty of room If there is even the slightest tendency to flat foot from laxity of muscles or overweight, small rubber sponge instep supports should be inserted, or the heels wedged  $\frac{1}{4}$  inch to  $\frac{3}{4}$  inch on the inner side, passing away to nothing on the outer side, like a wedge of cheese At least two pairs of shoes should be kept for each child, so that the feet can be rested

### SLEEP

**The normal child.**—The following table shows the amount of sleep required by children at various ages

TABLE III

Birth	20-21 hours
Six months	18
One year	14-16
Two years	12-14
Five "	10-12
Ten "	10-11
Sixteen,	9

In children up to the age of four or five it is well to introduce a middy rest Up to the age of four the rest is best given before dinner, say from 11 to 1, but between four and five it is best given after dinner, from 1.30 until 3

**Abnormalities in sleep.**—There are vast differences in the amount of sleep required by ordinary healthy infants Children who are played with and become too excited or who are allowed to be on their legs too much, sleep badly Digestive disturbances, too late supper and overfeeding the last thing at night tend to produce night terrors and disturbed sleep Tonsils and adenoids, blocking the airway, will produce broken sleep, at intervals throughout the night slow asphyxia occurs, with sudden bursts of crying in which the child recovers his oxygen Hunger pains and wind colic often cause disturbed sleep Many infants wake each time water is passed throughout the night and refuse to settle off again unless changed As they get older, and micturition is less frequent, the nights become better



*Feeding neuralgia* undoubtedly wakes some children as does the drooling or hypersecretion of saliva causing intermittent bursts of coughing.

In *encephalitis lethargica* sleep by day and wakefulness at night is common. In a child who has been perfectly normal and who after some febrile illness develops restlessness and wakefulness at night with a tendency to sleep during the daytime *encephalitis lethargica* may be suspected. *Tumours of the brain* especially *pineal tumours* are said to cause increased drowsiness but are very rare. In most febrile illnesses the child is drowsy.

### FRESH AIR, SUNSHINE AND EXERCISE

**Fresh air**—A London child requires from four to six hours' fresh air every day as a minimum for good health. Up to the age of four or five he should be out both morning and afternoon.

**Sunshine (artificial and natural)**—Natural sunshine is such a rarity in some quarters that the question of *artificial sunshine* has seriously arisen. On the whole however the value is overestimated and it is not necessary for a normal healthy child provided that small doses of cod or halibut liver oil are given daily. On the other hand it is useful in the treatment of rickets and in raising the immunity of children who are liable to catarrhs and those who suffer from flabbiness and watery tissues. Children with asthma also benefit. Undoubtedly after influenza and colds during the winter months a course of artificial sunshine is of value if well administered.

**Sun baths**—During the summer months whenever possible the child should be stripped and allowed to run and play with the sun shining directly on the body. This combined with sea air is of the utmost value to every normal child and especially to those who have been debilitated by rheumatism or tuberculosis. Exposure to natural sunlight just as to artificial sunlight should be gradual after a severe illness.

**Exercise**—While many children suffer from confinement and lack of fresh air very few children once they are able to walk suffer from lack of exercise. They manage to take the maximum amount which is good for them whether allowed out or kept indoors. On the whole little children tend to be too active and to take more exercise than is beneficial.

### DEVELOPMENTAL ABERRATIONS

There is quite a wide variation between the pathological and the normal particularly in growth in height and general development. Some families develop early whereas others mature late. The difference between two boys of the same age one an early and one a late developer is tremendous. No child therefore, should be termed pathological unless the variation is gross.

**Gigantism**.—This will be dealt with in detail on p. 367. Apart from the type which runs in families (hereditary) the vast majority of cases are due to an over activity of the anterior portion of the pituitary gland. An occasional case is caused by lack of development of the sex glands (hypogonadism) since their maturity is followed by the cessation of longitudinal growth.

In *precocious puberty* there is an abnormally early development of the

reproductive organs, and such children are remarkably overgrown at first. Tumours of the sex glands and suprarenals produce such a clinical picture (See p. 374).

**Infantilism.**—As a rule dwarfism accompanies infantilism. In *dwarfism* the defect is in and limited to the skeleton (Gardiner Hill). Dwarfism may be hereditary, i. e. smallness of stature may run in a family. Again, it may be due to some skeletal disease, such as achondroplasia, dyschondroplasia, or osteogenesis imperfecta. It may be due to acquired skeletal disease, such as rickets and spinal caries or spinal deformities such as follow infantile paralysis. Finally it may be due to hypergonadism, causing premature epiphyseal fusion, which is sometimes seen in precocious puberty.

In *infantilism* both the physical and mental make up of childhood persist into adult life, and the individuals remain small and under developed, with a disproportionately large head. The limbs are short, there is sexual hypofunction, and a childish mental outlook.

Some of the commoner causes of infantilism are chronic wasting disease, such as tuberculosis, and long continued starvation. This is often called "cachectic infantilism." Congenital heart disease or gross pulmonary disease may also be a cause. Sometimes the changes are specific at the growth cartilages, such as are found in congenital syphilis, scurvy, coeliac disease, rickets and renal dwarfism. Finally, infantilism may be due to an arrested development of the pituitary, the thyroid or the gonads (sex glands).

### THE THIN CHILD

Some children have been well nourished and then progressively lose weight, becoming thinner and thinner. Other children have always been thin and over active, coming of a thin stock.

The thin child must be carefully examined to exclude organic disease as a cause of wasting. Inadequate feeding, nervous over activity, prematurity, asthma, chronic naso-pharyngitis, tuberculosis, rheumatism, chronic indigestion, diabetes mellitus, and nephritis, may all produce progressive wasting, but a careful examination will exclude such diseases. A very rare condition, progressive hypodystrophy may occasionally be met. As a rule, it is the upper half of the child that is involved in this condition, and there is an almost entire absence of subcutaneous fat, the face, arms and chest presenting a picture of extreme emaciation.

The commonest type of thin child is the constitutionally thin (nervous-exhaustion) child. There is an ever increasing number of these thin children, who are the children of thin, over active, intelligent parents. The mother's complaint is that she "cannot get any flesh on him", he has "always been thin," although possibly quite a plump infant.

**Ætiology.**—Such a patient has a defective power of assimilating food especially fat, and tends from his nature to be over active and to expend too much energy. The result of this is that the balance between the intake of food and the output of energy is too nearly equal. An attempt to increase the intake by the addition of cream, milk, butter and eggs produces a bilious attack. These children suffer frequently from gastric or acidosis attacks (see *Cyclical Vomiting*, p. 123).

**Treatment.**—The problem reduces itself to cutting down the child's output of energy to the point where, with a moderate intake of food, he has a balance on the right side so that he thrives and gains weight. This can be done by limiting the length of his day and arranging for a midday rest. Little children of this type, up to the age of three or four, should be off their legs for half the day. School or kindergarten should be started early to enforce rest and discipline. There is no reasonable doubt that carbohydrate especially in the form of sugar is extremely well tolerated by such children and powdered glucose or plain sugar is a useful adjunct to their diet.

### OBESITY

It is exceedingly difficult to explain some cases of obesity. After encephalitis lethargica some children become extremely obese, and it is thought that there is a lesion in such cases in the region of the pituitary or corpora quadrigemina. After postnasc meningitis with hydrocephalus and in pituitary tumour undue obesity has been noted. Some Jewish children at the age of 8 to 12 tend to become extremely obese and to develop a picture very like Frohlich's syndrome. This seems to be a functional rather than an organic condition. The body seems able to absorb and store up fat but unable to call on it again. With this one way valve action when the fat pours in and is not burnt up some children are able to subsist on an extremely small intake. Actually, however a vicious circle is formed. Fat is laid on probably due to some metabolic error. Once having become fat the child is less active and being less active, tends to put on more fat. This vicious circle continues. With the increased weight there is a tendency for the arches of the feet to break down, and with sore feet there is even less inclination to activity.

**Distribution of fat.**—In the male child there is a tendency to a feminine type of distribution especially on the breasts. In the female much fat is laid on over the hips and shoulders. In about one third of the cases there has been obesity from birth, in one-third it occurs between the ages of 4 and 7 and in the remainder the onset is between 10 and 12 years of age. It is slightly more common in females than males. Out of 129 cases seen by the author 78 were girls and 50 were boys. Their ages varied from 5 to 17 years with the vast majority within the ages of 10 and 13 years. One or other of the parents of three fourths of the children was overweight, or had been so formerly.

There is not only an increase in weight, but the child is overgrown, being taller and broader than the normal for that age. There has been a definitely accelerated growth. As adults these children may not be abnormally large, in fact many of them are under average size. The result of this accelerated growth is that there is premature fusion of the epiphyses, as shown by X rays. Although the sexual characteristics are said to be retarded they are often normal for the age, though not for the physical development. The sexual organs may be obscured by rolls of fat, giving an erroneous impression of under development. Occasionally, however cases are seen where the penis and testicles are much under developed and the latter may be undescended.

**Treatment.**—A careful history should be taken to exclude organic

disease suggesting encephalitis, meningitis, disease of the thyroid or pituitary insufficiency

The diet should be regulated, and fat and carbohydrate reduced to a minimum. Very few eggs and from fifteen to twenty ounces of milk a day should be given, and there should be a great reduction in bread, cereals, and potato. The child should have a full allowance of meat, spinach, cabbage and lettuce. Sugar should be reduced to a minimum. Only three meals in the day should be allowed. Drinks of water need not be restricted. Fruit of all sorts may be given in moderate quantities in place of milk puddings.

#### SUGGESTED DIET FOR OVERWEIGHT CHILDREN

- \*Breakfast*
- 1 Raw fruit, orange, grapefruit, or apple. Stewed fruit (such as prunes, figs or apricots)
  - 2 Plasmon Oats or Energen cereal products, or Bemax. (Avoid cereals such as porridge, cream of wheat, puffed rice, cornflakes, etc.)
  - 3 Lean bacon, cold tongue, lean ham, fish, or a soft boiled egg (not more than three per week)
  - 4 Cellar's bread or biscuits or Energen wholemeal bread, Ryvita, Vita wheat or bran biscuits
  - 5 Plain water or freshly brewed tea (with very little sugar), orange or lemon water, or half a glass of milk
- Dinner*
- 1 A liberal quantity of red or white meat of any sort, or fish, may be given
  - 2 Fresh vegetables must be given in abundance, such as—lettuce, spinach, cabbage, cauliflower, asparagus, celery, onions, tomatoes, parsnips and carrots. Potato must be taken with great caution, try to manage without potato
  - 3 Fresh or stewed fruit—apples, pears, grapes, peaches, plums, cherries, pineapple and rhubarb—dried fruits—prunes, figs, raisins and dates. (Avoid milk and sweet puddings)
  - 4 Water to drink
- Tea*
- 1 Lettuce, tomato or watercress sandwich or Ryvita or Vita wheat spread with marmite or cream cheese
  - 2 Cup of tea or glass of milk
- Supper*
- 1 Salads or soups (excepting thickened and potato soups)
  - 2 A little fish (boiled, steamed or baked) or fish mayonnaise or cold tongue, ham or cheese
  - 3 One rusk or a small portion of Ryvita, Crispbread, Vita wheat or Energen wholemeal bread or bran biscuits
  - 4 Drinks as at breakfast

#### ARTICLES OF FOOD WHICH SHOULD BE GIVEN WITH CAUTION ON ACCOUNT OF THEIR TENDENCY TO FATTEN

- Sugar (including all sweets, chocolates, sweet jams, honey and syrups)
- Starchy Foods (such as porridge, bread, potato and rice pudding). Try to do without any bread or potato
- Fatty Foods (such as butter, cream and eggs)
- Drinks. Cocoa, Ovaltine, and dried milks should be avoided. Keep to plain water or weak tea, or cream and milk (a pint per day is required)

#### FOODS THAT ARE NOT FATTENING

- Meat, fish, soups (except thickened and potato soups), salads and green vegetables, fruit (raw and stewed) without much sugar
- Ryvita, Crispbread, Vita wheat, and Energen and Cellar's food products (with very little butter)

To ensure sufficient vitamins some cod or halibut liver oil preparation should be given three times daily at intervals.

*Exercise*—Massage, cold baths and vigorous exercise of all kinds should be persevered with. Swimming is especially important. Wedged shoes or shoes with instep supports may be necessary to correct a tendency to knock knees and flat foot.



Fig. 1—Case of obesity aged 11 years

*Medicinal treatment*  $\frac{1}{2}$  gram of thyroid gland twice daily may be safely given and if the child is under close medical observation this should be slowly increased up to  $1\frac{1}{2}$  or 2 grains a day. Symptoms of tachycardia and flushing should be watched for. Injections of 500 to 1 000 rot units of Pregnyl per week are said to have a most beneficial effect on those over weight children with undescended testes or in whom the testicles are under developed. It is necessary that the child should co-operate in every way with the dieting. No amount of urging on the part of the parent can control the situation. The diet therefore should be carefully explained to the child several times. Encouragement is afforded by giving the child access to an accurate pair of scales for daily use. When possible these should be placed in the bedroom.

**Prognosis**—Those cases in which no organic lesion or preceding disease can be ascertained and which are assumed to be functional tend to become normal, late in adolescence (Fig. 1).

### MORTALITY IN CHILDHOOD

Table IV shows the principal causes of death among children in England and Wales in the year 1942. In the first few months of life premature birth and congenital malformations make up a large proportion. The neo natal death rate is still extremely high. Infectious diseases cause an undue mortality under the age of 5 years but great strides are being made in their control.

The mortality in childhood generally is comparatively satisfactory however, as it shows a steady drop especially since the war of 1914-18. This may be attributed in part to additional knowledge of disease and its more efficient treatment but a very important factor is undoubtedly the improved social condition of the poor.

A standard which can be usefully applied to present conditions is furnished by the infant mortality from all causes for New Zealand during the period 1921-24 viz. 43.4 per thousand.

**Public Health.**—Students and practitioners who are taking the examinations in Public Health or the Diploma of Child Health are advised to read the following publications. The Health of the School Child. Annual Report of the Chief Medical Officer of the Board of Education for the year also recent numbers of *Archives of Diseases in Childhood*, *The British Journal of Children's Diseases*, *The American Journal of Diseases in Children*, *Journal of Paediatrics* and *Acta Paediatrica*. Garrod Batten, Thursfield and Paterson's *Diseases of Children* 4th edition Arnold 1945. Holt's *Diseases of Infancy and Childhood* by Holt and Mackintosh 11th edition, Appleton N.Y. 1940. Parson and Barling's *Diseases of Infancy and Childhood* Oxford Medical Publications 1933 and *Mother and Child* (The Official Organ of the National Baby Welfare Council and its Constituent Societies).

The author would also suggest that a model Welfare Centre, an open air school and a Day Nursery should be visited e.g. that on the former Foundling Hospital site, Mecklenburgh Square or the Welfare Centre and Day Nursery in Westminster or those of Poplar, Lambeth or other London districts.

TABLE IV.—DEATHS IN ENGLAND AND WALES 1913 (PROVISIONAL)

DISEASES	Total under 15 years		Under 1 year		1-5 years		5-10 years		10-15 years	
	No.	per cent	No.	per cent	No.	per cent	No.	per cent	No.	per cent
1 Measles	741	1.519	256	0.765	400	5.274	72	1.541	13	0.436
2 Scarlet fever	94	0.196	4	0.012	11	0.541	30	0.922	10	0.335
3 Whooping cough	1,111	2.319	621	1.957	157	6.026	33	0.916	—	—
4 Diphtheria	1,150	2.401	57	0.175	179	6.318	467	11.700	147	4.938
5 Influenza	637	1.373	337	1.008	191	2.518	57	1.160	72	2.417
6 Other infective and parasitic diseases (excluding the above)	818	1.770	736	1.065	29	3.517	142	3.837	101	3.392
7 Tuberculosis of nervous system	1,296	2.786	179	0.531	446	8.510	301	7.710	170	5.708
8 Tuberculosis of intestines and peritoneum	201	0.419	29	0.086	102	1.315	36	0.922	31	1.141
9 Other forms of tuberculosis	792	1.632	139	0.415	303	3.995	122	3.122	218	7.320
10 Syphilis	165	0.341	153	0.457	2	0.026	—	—	10	0.335
11 Rheumatic fever	233	0.486	1	0.003	17	0.221	100	2.561	115	3.862
12 Rickets	29	0.065	22	0.065	7	0.092	—	—	—	—
13. Meningitis	399	0.833	177	0.529	125	1.618	67	1.711	30	1.007
14. Convulsions	590	1.231	511	1.537	70	0.922	0	0.153	—	—

15	Other disease of nervous system and sense organs	1,177	2,457	522	1,561	254	3,349	210	5,379	191	6,410
16	Heart diseases	343	9710	7	0,021	26	0,342	88	2,252	222	7,454
17	Bronchitis	1,680	3,317	1,236	3,697	270	3,561	48	1,229	35	1,175
18	Pneumonia, all forms	7,416	15,546	5,693	17,029	1,118	18,670	202	5,174	133	4,466
19	Other diseases of digestive system	1,352	2,801	513	1,534	312	4,115	314	8,043	213	7,152
20	Diarrhoea and enteritis	3,027	7,572	3,354	10,032	230	3,033	23	0,589	20	0,671
21	Other disease of respiratory system	272	0,597	167	0,430	108	1,124	27	0,692	30	1,007
22	Disease of skin and cellular tissue	246	0,515	193	0,573	31	0,109	11	0,291	11	0,369
23	Congenital malformations	4,468	9,329	4,021	12,027	279	3,079	103	2,638	65	2,182
24	Congenital defects, etc.	853	1,789	853	2,552	—	—	—	—	—	—
25	Premature birth, injury at birth	10,334	21,375	10,334	30,941	—	—	—	—	—	—
26	Other disease of early infancy	2,443	5,161	2,443	7,306	—	—	—	—	—	—
27	Violence (all forms)	3,775	7,681	872	2,108	1,081	11,255	1,077	27,597	745	25,017
	Other causes	1,175	3,281	458	1,370	462	6,692	302	9,272	393	13,197
Total		47,896		33,431		7,583		3,904		2,978	



## CHAPTER II

### NORMAL BREAST-FEEDING AND BREAST-MILK

THE fact that breast feeding and breast milk suit the normal infant better than any possible artificial feeding is a truism which needs no stressing. A vast proportion of the mortality and morbidity in infants below the age of six months is to be found amongst those who are artificially fed. Most women can entirely or at least partially breast feed their children but at present<sup>1</sup> only just over 50 per cent suckle for more than three months.

By breast feeding an infant the mother saves the cost of the artificial food as well as much work and worry and it has the added advantage of aiding involution of the uterus. Her infant is five times less likely to die during the first year of its life if breast fed. The motions are better and the baby is plump and digestion is perfect. Probably, immune bodies are transferred from the mother to the infant in her breast milk.

The only disadvantage is that breast feeding takes a considerable amount of the mother's time but she must be prepared to sacrifice herself in this respect. Every woman cannot entirely breast feed her baby, and it is essential for the doctor to study the individual woman when advocating breast feeding. Some mothers will not make an attempt to breast feed at all. They also tend to wean for altogether inadequate reasons. It is the physician's duty to point out to everyone concerned the great advantages to the breast fed baby, and if necessary to insist on natural feeding.

**Preparation of the nipples**—The preparation of the nipples should commence two months before parturition. They should be cleansed frequently and kept well covered with cold cream. Yellow oxide of zinc ointment should be used to prevent cracks or sore tender or inflamed nipples. Harold Waller<sup>2</sup> contends that retracted nipples can be pulled out by gentle manipulation. He thinks that many difficulties can be avoided if the dried colostrum and secretions are expressed from the ducts for the month before the infant is born. He shows that the first milk from the breasts is thin and the late milk contains much fat. He emphasizes what is not sufficiently realised the force with which the milk flows or is ejected from the nipple at the time of feeding even before the infant is put to the breast. The mother may feel the draught or a tightness or tingling in the breast followed by the expulsion of the milk. This is a conditioned reflex and probably what happens is that the smooth muscle particularly around the large lactiferous ducts and also around the areola contracts causing this increased pressure in the breasts. Gunther suggests that the

Breast Feeding in Relation to Female Labour. *Arch Dis Child* 1943 xvi 1, 59

<sup>1</sup>Harold Waller, "Clinical Studies in Lactation" (Helsmann), 1937. "A Reflex Governing the Outflow of Milk from the Breast." *Lancet* Jan. 6 1943 i, 49

"draught" is accompanied by peristaltic waves passing down the ducts towards the nipple

**Colostrum.**—This is the fluid secreted by the breasts in the first few days after childbirth, and it is high in protein and low in fat and sugar content. It is possible that it conveys immune bodies to the infant but its exact significance is not understood.

**Breast-milk**—It will be seen from Table V that the fat content of breast milk varies from 3 per cent to 5 per cent. The globules, however, are extremely small compared with cow's milk and the emulsion is much finer. This makes its digestion much easier. The fat content of the milk

TABLE V (Holt)

SHOWING COMPOSITION OF COW'S AND HUMAN MILK AND VARIATIONS WHICH MAY OCCUR IN THE COMPOSITION OF BREAST MILK

Components	Cow's milk Per cent	Human milk Per cent	Common healthy variations in human milk Per cent
Water	86—87	88.05	87.82—89.60
Fat	4.00	3.70	3.00 — 5.00
Proteins	3.50	1.25	1.00—2.25
Milk sugar	4.50	7.00	6.00—7.00
Mineral salts	0.75	0.20	0.18—0.25

cannot be varied by giving a richer or poorer diet to the mother provided she is adequately fed. Protein is  $1\frac{1}{4}$  to 2 per cent, compared with  $8\frac{1}{4}$  to 4 per cent in cow's milk. The bulk of the protein is present as lactalbumin and lactoglobulin, and only a small proportion as casein. In cow's milk, on the other hand, the casein content is from four to five times as great as the albumin content. The clot formed in the stomach of the infant on breast milk is extremely small. By diluting cow's milk and therefore reducing the protein from 4 to 2 per cent, an attempt may be made to approximate cow's milk to breast milk but clearly the proportion of insoluble casein in cow's milk cannot be made to approximate to that of breast milk. Attempts at humanizing milk by dilution can never be strictly accurate. The carbohydrate present in breast milk is lactose. The amount present in breast milk is approximately 7 per cent which gives breast milk a much sweeter taste than cow's milk. The salts in breast milk are present in a much smaller quantity than in cow's milk. Unfortunately, at the present time, our knowledge of the role played by these salts is extremely elementary.

Infants feeding at the breast seem to have a much higher immunity to disease than those fed on artificial food. It is possible, therefore, that some immunity to diseases can be passed on from the mother to her infant through the breast milk, but of this there is no scientific proof. Noah Morris states that the sulphonamide group of drugs is excreted in breast milk. The author has not seen any ill effects on the breast fed infant as a result.

**Normal breast-feeding.**—Breast feeding suits nearly every infant

When on account of social duties or of temperament and general make up the mother is unable wholly to breast feed her child—and in some cases the longer the attempt is continued the more the child deteriorates—*complementary feeding* that is giving cow's milk at the end of the breast feed may be adopted with great benefit. The individual mother must be studied just as the individual child is studied in artificial feeding.

**Contra indications to breast feeding**—Where the mother has open tuberculosis she should not attempt breast feeding, as there is a risk of infecting the child. A syphilitic infant should be nursed by its own mother. In acute infections such as typhoid fever or pneumonia or puerperal septicæmia the mother is too ill to breast feed but in mild infections there is no contra indication. In heart disease nephritis, anæmia and epilepsy each individual case must be considered. The onset of menstruation during lactation should not be taken as an indication for weaning. The infant may require artificial food for 24 hours but should resume its breast feeds thereafter. The intervention of pregnancy during lactation is not in itself a contra indication and should the mother and the infant both thrive weaning is unnecessary. Should either however show signs of failing weaning should take place at once.

**Variation in quantity and quality of breast milk**—(see Table V, p. 23)—The first feed in the morning is probably the largest in quantity from that time the feeds get smaller and in the middle of the day reach their smallest remaining deficient until the evening when the amount again increases. The quantity of the milk seems to vary with the activity of the mother but the composition appears to be constant for any particular woman.

**Lactation and its establishment**—During the second 12 hours after the birth of the infant it should be put to the breast twice. *It is the suckling of the infant which stimulates the flow of milk and without this strong suction the establishment of lactation is impossible*—After the first 24 hours regular times of feeding should commence, three-hourly or four-hourly as the case may be and both breasts should be used at each feed.

**Complementary feeds and when they should be started**—Once the flow of milk is established both breasts can be used for each feed, the infant being kept at each for 7 or 8 minutes. Very often however, there is a delay in the establishment of the flow of milk, and during this time the child cries constantly is inclined to have loose stools and to swallow much wind, with the result that breast feeding is often abandoned. The weight of the child should be taken daily, and if the loss continues beyond the fourth day small complementary feeds should be given after each breast feed. This however, should be merely a temporary measure, to be adopted only until the flow of milk is fully established, when it can be discontinued. Such a complementary feed might consist of a table spoonful of boiled cow's milk and water with a quarter of a teaspoonful of sugar.

**Times of feeding**—The time and frequency of feeding must obviously depend on the size of the child and the supply of breast milk. If the child is small, naturally its stomach holds an insufficient quantity of milk to last it any length of time, and therefore frequent feeds will be necessary.

Thus, infants under 6 lb require at least seven feeds in 24 hours those from 6 to 8 lb in weight six feeds in 24 hours and those over 8 lb five feeds in 24 hours. For the average infant of 6 lb to 8 lb therefore, it will be advisable to feed at 6 a.m., 9 a.m., 12 p.m., 3 p.m., 6 p.m., and 10 p.m., whereas it is unwise to feed an infant over 8 lb at 6 a.m., 10 a.m., 2 p.m., 6 p.m., and 10 p.m. Should the supply of breast milk be too liberal even in a small baby the intervals between the feeds must be lengthened. *The times of feeding should be rigidly adhered to and the infant conscientiously awakened at these times.* The mother should be careful to compress the nipples slightly shortly after the feed commences to prevent the baby getting the milk too easily and gulping. The great bulk of the feed is obtained in the first five minutes and 15 minutes ought to be sufficient time for the whole. At the end the child should be wrapped tightly round in its shawl and placed upright against the mother's shoulder, to allow him to break the wind. This should invariably be done after each breast feed, as babies usually swallow wind, and an effort must be made to dislodge it, or discomfort and vomiting will result.

**Diet of the pregnant and nursing mother.**—The mother's diet should be the one which agrees with her best in normal times but in addition larger quantities of fluid, not necessarily milk, should be taken. If she can tolerate them, such sweet things as Horlick's Milk or Ovaltine, with a high sugar content, will be found to increase the flow of milk. It is quite wrong to supply the mother with gruel, stout and much milk, unless these suit her normally. Fresh fruit and vegetables are essential. In treating constipation it is best to avoid salines and calomel since they tend to lessen the quantity of milk. Senna, cascara or liquid paraffin will be found most efficient. Foods which are taken by the mother without ill effect do not seem to exert any ill effect upon the child. For instance, if the mother has eaten tomatoes or oranges or grapes and the following day a slight looseness appears in the infant's bowels there is no sound basis for attributing this to the mother's diet. Specifics disagreeing with the mother, however, are likely to upset the child.

Elbbs, Tisdall, Scott, Moyle and Bell<sup>1</sup>, who set out to determine the effect of poor and good pre-natal diets upon the outcome of pregnancy and condition of infants during the first months of life, advocated the following amounts of essential foods daily—40 oz. milk, 1 oz. cheese, 1 egg, an average serving of butter and meat, two servings of vegetables in addition to potato, 1 orange or half a grapefruit or 5 oz. of tomato juice, one half of the cereals and bread consumed to be in whole-grain form and two teaspoonsful of cod liver oil or equivalent in concentrate, liver once a week, salt to be iodized and medicinal iron to be used if indicated, two table-spoonsful of wheat germ daily also advised.

Capsules and tablets containing 4,000 I.U. of vitamin A and 800 I.U. of vitamin D have been made available by the Government to expectant mothers.

**Failure to breast-feed.**—This may be due to a defect in the child or to a defect in the mother.

<sup>1</sup> *Journ. Nutrit.*, 1911, XXII, 515.

*Canad. Med. Ass. Journ.*, 1914, XLVI (1), 4.

1 Defect in the child—Anything which interferes with the suction of the infant at the breast will cause a failure in lactation. Nasal obstruction due to catarrh or adenoids and harelip with cleft palate cause great difficulty in sucking. Sometimes the infant is premature and under weight and not strong enough to suck properly. Or it may have congenital heart disease or atelectasis of the lung. In these cases the milk may have to be pumped off or expressed and the feed given with a spoon or bottle. Some infants are poor suckers the co-ordination between sucking breathing and swallowing being badly developed. Success in such cases depends on the energy with which the situation is handled.

2 Defect in the mother arises usually from one or more of the following conditions—

- (i) Worry or mental upset this is the most potent cause. If she is overworked and undernourished the supply of breast milk will be deficient.
- (ii) Failure on the part of the mother, nurse and physician to persevere in getting the infant to take the breast in the first fortnight after birth.
- (iii) Breast infections (abscess cracked or sore nipples)
- (iv) Malformation of the nipples that is depressed nipples, or poorly developed breasts.

When the breast has become infected so that there are tender areas or actual abscess formation breast feeding may have to be discontinued entirely since the general upset to the mother is considerable. In such circumstances the milk should be expressed or gently pumped off to relieve the mother and the child fed on an artificial feed or given milk from the uninfected breast with a complementary feed of cow's milk and water.

**Methods of increasing the flow of breast-milk**—Once lactation has been established it is maintained by the strong suction of the infant emptying the mother's breasts at regular intervals. This is the most potent factor in the establishment and maintenance of lactation. The supply of breast milk may be increased by

- 1 Improving the eating and sleeping habits of the mother.
- 2 Seeing that she has a sufficient fluid intake and especially that she is having some of the malted foods. So called galactagogues do not appear to increase the flow of breast milk.
- 3 Making certain that the breast is completely emptied at each feed by pumping off or expressing the last drops of the breast contents.
- 4 Inducing a feeling of confidence in her ability to nurse her child successfully. This may demand an exaggerated air of optimism on the part of the doctor and nurse.
- 5 Breast massage and the application of hot and cold douches to the breast.

**Underfeeding on the breast**—Authorities differ whether underfeeding or overfeeding is the more common in breast fed babies. There can be very little doubt that of those breast fed infants who are brought for medical advice the vast majority are underfed.<sup>1</sup> The complaint is

<sup>1</sup> Donald Laterson and A. Murr-Collins, *The Frequent Occurrence of Underfeeding in Early Infancy* Arch. Dis. Child., No 11 Oct. 1937 p 215

often merely that the child is failing to gain in weight, at other times that it is restless during the day and sleepless at night. The stools may be constipated when the supply is only very slightly deficient, but later they are small, slightly greenish and often frequent—so called "hunger stools." Vomiting may also be a symptom. The child, put to the breast in a ravenous state gulps what little milk there is, and with it much air as the wind is returned some of the milk is returned with it, and thus further reduces the already meagre supply of milk so that at the next feed an even more ravenous infant gulps wind. It is this picture which has given rise to the expression "windy nuck" though probably no such thing as "windy milk" exists, really the milk is insufficient. An infant that is underfed cries a great deal especially before its feeds are due. It also cries after being fed having come from swallowed wind. Thus crying gives the impression that the child has indigestion and the parents think the breast milk is too rich or that the child is getting too much and consequently water is often given before the feed to dilute the strength. If the infant is truly underfed, its condition will be aggravated by such treatment.

**Diagnosis of underfeeding.**—To ascertain whether an infant is underfed, a series of "test feeds" should be given, that is the infant should be weighed carefully, either naked or in its clothes or shawl then put to the breast, then reweighed, and the difference which is the amount of milk obtained, carefully noted. A series of these tests taken over 24 hours is necessary in order to come to any proper conclusion as the feeds vary in size. A mother often insists that she has plenty of breast milk and that it flows away from her. The size of the breast its tension and the discomfort of the mother are believed to be signs of the amount of milk available, but none of them can be relied upon, the only certain criterion being a test feed.

**Amount of breast-milk required in the day.**—To judge the efficiency of breast feeding it is necessary to know how much breast milk a normal healthy infant requires to ensure that it will thrive. This is from  $2\frac{1}{2}$  oz to  $2\frac{1}{2}$  oz for each of the baby's pounds in the day, that is, a 6 lb infant requires  $6 \times 2\frac{1}{2} = 15$  oz of breast milk in the day. A 10 lb infant requires  $10 \times 2\frac{1}{2} = 25$  oz of breast milk in the day. It is easily seen, then, that one isolated test feed is of no value but it must be the amount of breast milk given during a particular day that is ascertained. Each infant is a law unto itself, some infants require slightly more food than others and nervous, highly strung infants especially require more food than placid infants who sleep much. In cold weather more food is required than in hot, sultry weather, and therefore the figure given above is only an average. It should be clearly understood, however, that the figure of  $2\frac{1}{2}$  oz of breast milk per pound of body weight per day applies to the normal healthy infant. Once an infant is underweight the calculation should be made on the *expected weight* rather than on the real weight. For example, a normal infant is born weighing  $7\frac{1}{2}$  lb, through some accident the weight decreases until at one month the weight is only 7 lb. The expected weight is 9 lb and the calculation should be  $9 \times 2\frac{1}{2} = 22$  to 23 oz in the day.

- 3 Colic, restlessness, disturbed sleep, failure to gain in weight. Very often the overfeeding is due to too frequent feeding, or leaving the child at the breast for too long when there is an over abundant supply of breast milk.

#### Treatment

- 1 Test feeds should be made to ascertain the amount of breast milk
- 2 Only one breast should be given, instead of both
- 3 The periods between the feeds should be lengthened from three hourly to four hourly intervals and no night feeds should be given
- 4 The time at the breast should be shortened
- 5 A little water may be given before the breast feed

**Weaning and the commencement of mixed feeds.**—Weaning should take place by degrees, thus the infant is not upset and the mother's milk is allowed to disappear gradually preventing painful breasts. A period of cold rather than warm weather should be chosen. There is no set age at which weaning should take place. When an infant weighs 15 lb it will be getting about 85 oz. of breast milk from its mother and it is seldom that the average woman can produce more than this without detriment to her own health. The solution of the difficulty is to give the child something besides breast milk. *Mixed feeding* should commence at this point, usually about the fifth or sixth month. The first stage is to give one to two teaspoonful increasing to two tablespoonful of bone and vegetable broth (see p. 30) at the 2 p.m. feed. This may be liberally thickened with potato and finely pureed vegetables a little later. The addition of Nestlé's, Heinz, Brand's or Libby's homogenized tinned vegetables would be suitable at this age. The second stage is to give a feed of groats,  $\frac{1}{4}$  or  $\frac{1}{2}$  teacupful, made with milk, at the 10 a.m. feed. This may be given from a bottle or, better, from a spoon. At 6 p.m. one third to half a teacupful of some starchy preparation such as Groult's Cream of Rice, Robb's Discuits Neave's, Sister Laura's or Ridgway's Food, or Allenbury's No. 9, or Glaxo Malted Food, M.O.F., or Farax, may be given as the third stage. The fourth stage is the addition of one to two teaspoonful of the yolk of a soft boiled egg to the groats at 10 a.m. Rusk should be given as soon as the lower incisors appear. Each of these stages should occupy about a week.

#### DIET FOR A NORMAL HEALTHY BREAST FED INFANT FROM FIVE TO NINE MONTHS OLD (WEIGHT 15 TO 18 LB.)

*Feeding Times* 6 a.m., 10 a.m., 2 p.m., 6 p.m., 10 p.m.

6 a.m. Give both breasts, 7 minutes to each side

10 a.m.	1	1	2						
		1	ounce						
		1	ounce						
		1	level	teaspoonful					

To this add one to three heaped teaspoonful of Chapman's Entire Wheat Food, Robinson's Patent Groats or Barley, Groult's Cream of Rice, M.O.F., Neave's Food or Sister Laura's Food. Half a teaspoonful of the yolk of a lightly boiled egg, should be slowly introduced along with this feed and gradually increased to two teaspoonful if well tolerated. (See below, Note on cooking)

- 2 After this feed give the breast

2 p.m. 1 Milk mixture as at 10 a.m.

Add to this 2 tablespoonsful of bone and vegetable broth (*see below*)  
One or two tablespoonsful of Heinz Libby's, Brand's or Nestle's homogenized vegetables may be added with advantage

2 Give the breast

6 p.m. 1 Give the feed exactly as at 10 a.m. but add one to three heaped teaspoonsful of a different cereal, and omit egg

2 Give the breast

10 p.m. Give the breast only as at 6 a.m.

**Fruit Juice**—Orange or tomato juice, two to three teaspoonsful, diluted with water and sweetened with sugar or a similar quantity of black currant purée or rose-hip syrup diluted with water should be given daily. Should none of these be obtainable the alternative is 50 mgm. of ascorbic acid. A convenient time for this is between 8 and 10 a.m. or at tea time.

**To I rectal Pickets**—Give one third of a teaspoonful of Government vitaminized cod liver oil immediately before three of the feeds, or one drop of halibut liver oil.

**Note on Cooking**—All milk should be brought to the boil. The cereal, such as Groats or Cream of Rice given at the 10 a.m. and 6 p.m. feeds, needs to be cooked directly for at least 10 minutes or from half an hour to an hour in a double saucepan. It may be added to the rest of the feed and the whole cooked for the specified time or it may be cooked with water and then stirred into the rest of the feed when it is thoroughly cooked.

**✓ L**—This feed is so thick that a large hole must be made in the teat if given from a bottle but if possible it is better to spoon feed from a cup.

#### Bone and Vegetable Broth

Take 1 lb. of veal or beef bones or calves' feet, well broken up. (During war time, chicken or rabbit bones may be substituted, or, where bones are not available, a small portion of meat such as beef, veal or lamb, cut into small pieces). Cover with water and add one teaspoonful of vinegar. Occasionally say once a fortnight add a small piece of calves' or ox liver (about 2 ozs.) Simmer for from four to seven hours. Now add vegetables (carrots, cauliflower, swedes, parsnips, green vegetables and potatoes). Simmer for one more hour strain and allow to set. The broth is best cooked in a double saucepan, and should keep for three days in a cool place.

**✓ B**—A similar broth can be purchased "ready made" from Bickings Ltd., Nursery Food Specialists, Dept. 12, Welwyn Garden City, Herts. (Telephone Welwyn Garden 428).

At about nine months bottles may be substituted for the breast, one feed at a time, and it will be found suitable to give a 7-oz. feed composed of 5 oz. of cow's milk, 2 oz. of water and a heaped teaspoonful of sugar (*see p. 44*). Should the mother complain, the breasts may be relieved by support with a bandage. Her bowels should be kept well open with aperients at this time, and the breasts should be emptied at bedtime by expressing.



## CHAPTER III

### THE ARTIFICIAL FEEDING OF THE NORMAL INFANT AND CHILD<sup>1</sup>

EVERY infant should be breast fed wholly or at least in part. Artificial feeding is a procedure which necessity alone forces on the infant. Where for some reason, breast feeding cannot be carried out certain principles of artificial feeding must be considered. The importance of understanding the fundamentals of infant feeding is being more and more appreciated both by the doctor and the general public. No practitioner can be said to be a properly trained doctor without a full knowledge of these principles.

TABLE VI  
COMPOSITION OF MILK FROM DIFFERENT MAMMALS  
(Modified from Abt's Pediatrics)

Mammal	Specific Gravity	Water	Casein	Albumin	Total Protein	Fat	Sugar	Ash	Total Solids
Man	1.024	87.3%	0.40	1.21	0.61	3.4	6.3	0.0	10.42
Cow	1.0313	87.2%	2.88	0.51	3.31	3.78	4.74	0.72	14.5
Ass	1.0250	90.2%	0.2	1.70	1.55	1.3	8.19	0.17	9.88
Goat	1.020	86.8%	.47	0.45	3.76	4	4.04	0.84	13.12
Mare	1.034	90.85	1.50	0.75	2.0	1.11	5.8	0.36	8.4

#### COW'S MILK

**Graded milks**—1 Tuberculin tested milk is produced from cows which have passed the tuberculin test and a veterinary examination. These cows are tested and examined at intervals of three months. The milk is bottled on the farm and must pass the new methylene blue reduction test for its bacterial content. This may be pasteurized and will then be called Tuberculin tested (pasteurized), if bottled on the farm it will be called Tuberculin tested (certified).

2 Accredited milk is similar to No. 1, but the tuberculin test is not required. A veterinary examination of the herd takes place every three months. This milk will be subjected to the same bacteriological tests (methylene blue) as No. 1.

3 Pasteurized milk is milk which has been kept at a temperature of 145°-150° F for 30 minutes. Milk so treated gives protection against milk borne disease.

*Is it necessary to boil cow's milk?*—By boiling cow's milk the curd is made more digestible. It is true that the vitamins are slightly diminished in the process though not destroyed but these are easily made good by giving fresh fruit juice and a little cod liver oil, the latter especially during the winter months. The chief advantage of bringing milk to the boil is

<sup>1</sup> Most of the tables in this chapter have already appeared in Patterson and Forest Smith's "Modern Methods of Feeding in Infancy and Childhood" (Houstable & Co. Ltd.), where the whole subject of infant and child feeding is dealt with in much greater detail.

that all pathogenic organisms are killed off. The commonest and most to be feared is the tubercle bacillus, but epidemics of diphtheria, scarlet fever and infantile paralysis have all been traced to the milk supply, and the simple precaution of scalding the milk prevents infection from this source. The author considers that all milk given to a child should be boiled or pasteurized.<sup>1</sup> Tuberculin tested certified pasteurized milk is the cleanest and safest milk obtainable.

**Condensed (evaporated) milks**—These milks are manufactured by heating milk and evaporating it in vacuo. Once a tin of the unsweetened variety has been opened it will not keep longer than fresh cow's milk. The sweetened varieties, however, because of their high sugar content, will keep considerably longer.

The composition of a number of the better known brands of condensed milk is given in Appendix II.

**Sweetened full cream condensed (evaporated) milks**—The composition of this group is roughly fat 10 per cent, protein 9 per cent, carbohydrate 54 per cent. When reconstituted, by adding one teaspoonful to an ounce of water the composition becomes fat 1.2 per cent, protein 1.1 per cent and carbohydrate 6.8 per cent. (For detailed table, see p. 89.) In this group the vitamins have been almost entirely conserved and the milk is sterile. The tendency in making up such milks, which have added sucrose is for the protein and fat content to be low compared with the high carbohydrate content. A little fresh cow's milk and cod liver oil emulsion should be added to the diet of infants fed on these milks which then form a satisfactory food for the normal infant.

**Unsweetened full-cream condensed (evaporated) milks**—The composition of this group is roughly fat 9.5 per cent, protein 9 per cent and carbohydrate 12 per cent. When reconstituted by adding one part of evaporated milk to three parts of water, the feed would be fat 2.5 per cent, protein 2.2 per cent and carbohydrate 3 per cent. To such a feed, sugar would need to be added in the proportion of one level teaspoonful to each 4 ounces of the mixture.

After one month the mixture could well be strengthened to one part of evaporated milk to two parts of water, with one level teaspoonful of sugar added to each 2½ oz. of the mixture. This would give a composition of fat 3.2 per cent, protein 3 per cent and carbohydrate 9 per cent. This could be considered a satisfactory mixture to feed a normal infant. After six months a mixture of equal parts of condensed (evaporated) milk and water replaces cow's milk in the dietary. Such a method of feeding has been used of recent years both in the U.S.A. and Canada, with great success. Both fresh orange or tomato juice, and cod or halibut liver oil, are required to complete the feed.

**Sweetened skimmed condensed (evaporated) milks**—Such condensed milks are unsuitable for the human infant without some additions, but if cow's milk and cod liver oil emulsion are also given their deficiencies

<sup>1</sup> Reports on the Supervision of Milk Pasteurizing Plants, by Sir Weldon Dalrymple Chambers, Report No. 77 H.M. Stationery Office 1922.

C. F. Brockington, The Evidence for Compulsory Pasteurization of Milk, *Brit. Med. Journ.*, March 27 1927 166.

G. B. Wilson, The Pasteurization of Milk, London, 1927 Edward Arnold.

are made good. Fruit juice should always be given. Older children who are thin and under weight, and have a poor tolerance for fat, sometimes thrive on such milks, spread thickly on bread and butter in place of jam.

**Dried milks.**—During the process of drying cow's milk the curd is modified to some extent, so that it becomes more easily digested by the infant than that of fresh cow's milk. Dried milks are made in two ways either by pouring fresh milk on hot rollers, or by the spray process in which it is dried by being forced through small jets into a hot atmosphere.

*The advantages of dried milk are:* (1) It is sterile and tends to remain so over long periods. (2) in the process of drying the curd has been modified and becomes more digestible than in fresh milk. (3) it is easy to keep a supply, and this is a great advantage when travelling or when fresh milk cannot be properly kept, (4) the vitamin loss is very small. moreover this point is becoming less important, as cod liver oil and fruit juice can be given as a safeguard.

**Full-cream national dried milk.**—The disadvantage of dried milk is the cost, which always exceeds that of fresh milk. For practical purposes dried milks are made so that 1 part dissolved in 8 parts of water reconstitutes whole cow's milk. Therefore, a heaped teaspoonful dissolved in 1 oz. of water reconstitutes an ounce of cow's milk.

The composition of an average good brand of full cream dried milk is roughly: protein 26 per cent, fat 26 per cent, carbohydrate 38 per cent. If such a dried milk be reconstituted by adding 1 heaped teaspoonful to 1 oz. of water, the result will be: protein 8.2 per cent, fat 8.2 per cent, carbohydrate 4.7 per cent. It is advantageous, therefore, to add 1 level teaspoonful of sugar to each 2½ oz. of the mixture, as otherwise the carbohydrate will be unduly low, and the feed badly balanced. For full table of dried milks, see Appendix II.

**Humanized dried milks.**—These are made so that one teaspoonful dissolved in 1 oz. of water reconstitutes an ounce of breast milk, as nearly as possible. The composition of a typical humanized dried milk is roughly: protein 14 per cent, fat 25 per cent, carbohydrates 55 per cent. When reconstituted by adding 1 heaped teaspoonful to 1 oz. of water, the percentage composition becomes: protein 1.8 per cent, fat 3 per cent, carbohydrate 7.9 per cent. This is a suitable food for infants during their early months. Cod or halibut liver oil and fruit juice, are of course necessary in addition. For full table see Appendix II, p. 401.

**Starchy proprietary foods.**—The majority of the starchy proprietary foods are made up by adding them to cow's milk. Some require cooking while others such as Savory & Moore's Food, or Benger's Food, Pablum or Larex need only be added to the water milk mixture and allowed to stand. Such proprietary foods are extremely useful to introduce starch into the diet of the infant after the fifth or sixth month, and they may also be used in the early months of life to thicken the food of babies addicted to rumination (see p. 117).

The average composition of the starchy preparations (for list, see Appendix, p. 102) shows: protein 5 to 10 per cent, fats 2 to 10 per cent, carbohydrate 70 to 80 per cent. One to three heaped teaspoonful would be sufficient to add to the single 7 oz. feed of an infant.

When a proprietary food is used the greatest care must be taken not to give excess. If the proportion of starch be too high, the infant becomes flabby and pale with a low resistance to infection. Many of the proprietary foods are deficient in vitamins and therefore half a teaspoonful of cod liver oil or 1 drop of halibut liver oil should be given three times daily and fruit juice 3 to 4 teaspoonful daily should invariably be added.

The baby who will not tolerate sugar given as cane sugar or lactose will often take an adequate amount of carbohydrate in a preparation containing a mixture of dextrins and maltose (partly digested starch) or again may fail to gain weight on simple milk mixtures and will thrive at once when a more starchy food is added to the diet. At the period of weaning a proprietary food may initiate with success the first attempt to give more than milk.

#### DIFFERENCES BETWEEN COW'S MILK AND BREAST MILK

There are fundamental differences between cow's milk and breast milk which can never be overcome. Breast milk is received by the infant warm and sterile while cow's milk reaches the infant after some hours and has always been contaminated. The protein and fat of cow's milk can never be made similar to that of breast milk.

The main differences in composition between cow's milk and human milk have already been set out in Table V, p. 23. The question arises whether it is necessary in artificial feeding that cow's milk should be modified to become as nearly like breast milk as possible. The answer is that since we cannot produce a mixture like breast milk we should concentrate on producing a mixture on which the infant thrives as well as on breast milk. This may necessitate departing widely from the breast milk standard. Cow's milk is composed of protein (casein and lactalbumin), fat, carbohydrates and salts.

**Protein**—When the protein of cow's milk reaches the stomach it is acted on by gastric secretions producing a clot. It is casein which clots, becoming calcium caseinate. These curds may be made smaller or modified by the following means:

1. Boiling the milk precipitates the casein so that the curd is small when formed in the stomach. Pasteurization does not inhibit curd formation.
2. Diluting the milk with water causes smaller curds which are more easily digested.
3. Dilution with cereal waters. When the milk is diluted with a colloidal solution of barley or oat starch the particles of casein seem to be held apart and the curds remain extremely small.
4. By peptonizing. The curd may be almost completely obliterated in this way, but the method is rather complicated and expensive for universal use.
5. By the use of dried and condensed milks. In the process of drying and condensing milks a change takes place in the protein so that the curds are distinctly smaller than in fresh cow's milk.
6. By the use of alkalis—sodium bicarbonate, sodium citrate, calcium hydroxide (lime water) or magnesium hydroxide (milk of magnesia).

Possibly these join up with the casein to form smaller molecules but in any case the curd formation is much smaller when they are added

- 7 By adding dilute hydrochloric acid (B P) to the milk in the proportion of one and one-half drops for each ounce of cow's milk in the mixture, the "buffer" of the milk is filled. The natural gastric juices are in this way freed to raise the feed to the optimum acidity for gastric digestion

If there is an excess of protein the infant will fail to gain weight and tend to be constipated, since alkaline constipated stools are produced by an excess of protein. Protein curds are seldom seen; they are large bean-shaped, brown and semi-translucent. In underfeeding on protein the infant tends to be progressively anæmic and rachitic.

**Fat.**—This is probably the most difficult element to digest in cow's milk. The globules are large and split down into fatty acids, which some infants are unable to assimilate. The stomach empties more slowly on high fat foods, and in warm weather there is a tendency to diarrhoea. There is no method of modifying the fat of cow's milk.

**Carbohydrate Sugar.**—This is present as lactose or milk sugar. Sugar is essential if a child is to gain weight but very few infants can manage more than two ounces (8 heaped teaspoon-ful) of sugar added to their daily feed. The metabolism of fat requires sugar to prevent ketosis (acidosis). The most easily assimilated sugar is dextrin maltose not lactose as is commonly supposed.

**Excessive sugar causes**

- 1 A very rapid gain in weight, the infant retaining much fluid in its tissues and becoming extremely watery
- 2 A tendency to loose, frothy, acid stools, produced by the sugar breaking down into the lower fatty acids

**Insufficient sugar causes**

- 1 Failure to gain in weight, constipation
- 2 Chronic biliousness and acidosis, with a dirty tongue

The aim in infant feeding is to get a nice balance between the alkaline producing protein and the acid-producing fat and sugar. The constipated child who fails to gain weight rapidly is an invitation to add more sugar to the diet. For practical purposes ordinary Demerara or cane sugar is quite sufficient in feeding the average healthy infant. Where there is a poor tolerance for sugar, however, dextrin maltose preparations such as Mead's dextrin maltose or Mellin's Food, are better.

**Starch.**—When an infant reaches the weight of about 15 lb. the carbohydrate must be increased along with the rest of its food, to keep the diet balanced. The average infant, however, is not able to manage large quantities of carbohydrate in the form of sugar, and starches should be commenced at this time. The starch, in process of digestion, is split into sugar, but since this process is slow, fermentation is not so likely to occur. Many infants who fail to gain when sugar is added to their feed will gain rapidly when starch is substituted for a portion of the sugar. It is due to this fact that a number of the well known starchy infant foods have gained their deserved reputations (see Appendix II, p. 402).

**Water.**—Diluting the feed makes it more digestible and supplies fluid which is utilized in metabolism. In summer more water is required. If water is withheld the infant becomes dehydrated, the urine is concentrated and scanty and the temperature rises (see pp 57, 74 Dehydration fever). Water given in addition to breast feeds helps to correct constipation.

### METHODS OF ARTIFICIAL FEEDING

1 **Undiluted cow's milk**—This will be found unsuitable in practice except in some few cases. The majority of infants will tolerate it but it is not on the whole a desirable.

2 **Humanized cow's milk mixture**—To make up a mixture roughly similar in composition to that of human milk is undoubtedly attractive. As the infant requires more and more food this mixture is increased in amount only but not in strength. Some small healthy infants thrive well on this but there are a large number who find the quantity of sugar too great and develop diarrhoea and flatulence. This is easily explained as the total amount of sugar offered to the infant in the day when very young is but small. As the child increases in size and its demand for food increases the total quantity of sugar offered in the day increases rapidly and when the amount of added sugar in the day amounts to more than about 2 oz diarrhoea frequently results especially in warm weather.

3 **Simple dilution of milk, with the addition of carbohydrate.**  
**Method of choice**—The first step is to think in quantities necessary for 24 hours rather than in amounts necessary for individual feeds. The expected weight of the infant is much more help in calculating its food requirements than is its age and while it may not lead to an absolutely accurate result it is the most useful basis we have at present. The individual infant is a law unto itself. No rule can be set out which covers every individual case.

#### EXPECTED WEIGHT

<i>Method of Calculation</i>	<i>Example</i>
Birth weight	Birth weight 7 lbs 3 1/4 oz
Plus 1 oz per day of life (excluding first 10 days) for 100 days.	Age 40 days
Thereafter the infant should gain 1 lb per month up to the age of one year	Therefore a 1d 40 ozs (i.e. 10 ozs) i.e. 30 ozs
	1 lb 14 ozs
	<hr/> 9 lb 1 oz

**Calculation of the food requirements of infants**<sup>1</sup>—A breast fed baby requires 2½ oz of milk per pound body weight per day. It seems rational, therefore, that the artificial feed should be made up to the same quantity, that is a 10 lb baby requires  $10 \times 2\frac{1}{2} = 25$  oz of fluid in the day. The food of an underweight or overweight infant should be calculated on the expected weight rather than the actual weight at the moment. For example, a 7 lb infant who has remained (although 2 months old) at its birth weight, should have gained 9 lb in this period. It should, therefore, be fed as if weighing 10 lb and not 7 lb.

**Cow's milk requirements**—The milk should always be boiled, and an infant requires 1½ oz of cow's milk for each of its pounds in the day. Thus an 8 lb baby requires  $8 \times 1\frac{1}{2} = 12$  oz of fluid in the day, of this  $8 \times 1\frac{1}{2}$

<sup>1</sup>Donald Paterson and Ruth D'aly. A Study in Infant Feeding—Food Requirements of Infants. *Lancet* Jan 31 1926 1 237

= 14 oz should be cow's milk and therefore 6 oz of water will be added. Sugar of course must be added to this (see p 35), and half a teaspoonful of cod liver oil or one drop of habbut liver oil should be given three times daily. Put another way, the child requires a feed containing approximately two parts of cow's milk to one of water. 2½ oz of this mixture together with a level teaspoonful of sugar for each pound of the body weight in the day.

*Full cream dried milk* (Cow and Gate, Glaxo, Dorsell's Ambrosia Lacta see Table p 400) — One very heaped teaspoonful or measure of any of the above dissolved in an ounce of water reconstitutes one ounce of cow's milk. The infant requires 1½ measures or teaspoonsful for each of its pounds in the day, that is a 7 lb infant requires 11 or 12 heaped teaspoonsful or measures of Glaxo spread over the day. The amount of water this would be dissolved in would, of course, be that quantity which it would get if it were being breast fed, namely 2½ oz per pound body weight per day. An example is an 8 lb infant fed on Cow and Gate who would get 14 heaped teaspoonsful or measures of Cow and Gate dissolved in  $8 \times 2\frac{1}{2} = 20$  oz of water in the day. Sugar, 8 level teaspoonsful must of course be added to this feed.

**Sugar** — Sugar must be added to both ordinary cow's milk feeds and full cream dried milk. It is a very useful rule to add one level teaspoonful for each of the infant's pounds in the day, that is to the day's feed for a baby of 7 lb 7 level teaspoonsful should be added. To the 10 lb infant's feed 10 level teaspoonsful should be added. This infant however is fed five times in the day, so to each of his feeds two level teaspoonsful or one heaped teaspoonful should be added. One lump of Tate and Lyle's white sugar weighs almost exactly 60 grains and is the equivalent of a level teaspoonful of sugar.

Lactose is not the sugar of choice. The most easily absorbed sugar is dextrin maltose, a cheap form of which is Demerara sugar. The better forms are Mead's Dextrin maltose, Wander's Nutromalt, and Mellin's Food.

*Humanized dried milks* (Humanized Glaxo Allenburys 1 and 2 Humanized Trufood, Humanized Cow and Gate see table, p 401)

The essential thing about the humanized dried milks is that one very heaped teaspoonful or measure dissolved in 1 oz of water reconstitutes as nearly as possible 1 oz of breast milk. In calculating the food requirements with these humanized dried milks, one would give 2½ heaped teaspoonsful or measures dissolved in 2½ oz of water for each of the baby's pounds in the day. No sugar need be added. Often it is an advantage, for new born infants or premature infants to add one drop of dilute hydrochloric acid (B.P.) for each measure of the dried milk.

The greatest use for humanized dried milks is in the first few weeks of life. Certainly after the third month it is desirable to increase the proportion of curd as the sugar is increased, and the change from humanized milks to full cream mixtures at about this period is customary.

**Unsweetened condensed (evaporated) milk** (Ideal, Libby's Carnation) — A dilution of one part of the milk to three parts of water with added sugar, is required at birth (see p 399), but at one month the mixture is strengthened. Three-quarters of an ounce of condensed (evaporated) milk, together with 1½ oz of water, and 1 level teaspoonful of

sugar per pound body weight per day, is required. This will be found an extremely satisfactory method of infant feeding and is much used on the American continent. (For table see p. 43)

### CHOICE OF FOOD

In choosing a food for an infant various considerations arise. It may be that there is travelling to be done in which case a humanized or full-cream dried milk might be more suitable. It may be impossible to obtain a reliable fresh milk and cost might be of paramount importance. When circumstances permit however it is probably best to use fresh cow's milk making up the whole 24 hours' food in Soxhlet bottles and sterilizing it all at the same time. Then the individual feed only needs warming up. A resume of the advantages and disadvantages is given below.



Fig. 2.—Soxhlet bottle and an ordinary boat-shaped bottle

- 1 Unboiled cow's milk (not recommended)  
*Advantages*—Unaltered taste vitamins not destroyed simple to prepare and cheap  
*Disadvantages*—Not sterile not very digestible
- 2 Boiled cow's milk  
*Advantages*—Cheap digestible, sterile  
*Disadvantages*—Altered taste partial destruction of vitamins (these are however easily made up with cod or halibut liver oil and fruit juice)
- 3 Dried milks (Full-cream Glaxo Cow and Gate)  
*Advantages*—Sterile constant in quality, easily digested easy to obtain keep and make up  
*Disadvantages*—Expensive slight deficiency of vitamins which are easily made up with cod or halibut liver oil and fruit juice.



- 4 Humanized dried milks (Humanized Glaxo Humanized Cow and Gate Humanized Trafood Allentury's 1 and 2 etc.)

*Advantages and disadvantages the same as those of full cream dried milks*

**Choice of bottle**—There are two types of bottle on the market—the boat shaped bottle such as the Allenburys or Glaxo and the upright or Soxhlet type (Fig 2) with an opening at one end only. This latter has the advantage that it can be sterilized as it is stood on end but it cannot be kept clean as easily as the boat shaped bottle. The hole in the teat should be on the large rather than on the small side as with too small a hole the infant becomes tired and tends to suck wind. In any case the teat should be withdrawn from the infant's mouth at very frequent intervals to allow air to enter the bottle and thus prevent the teat from flattening out in the mouth. The best method of making a hole in the teat is to drive the red hot point of a needle through it. The temperature of the feed should be about 100° F. The author prefers a boat shaped bottle and when the feed commences the valve should be removed so that the teat need not be removed from the infant's mouth. Whichever type of bottle is used it will be found most convenient to make up the total feed for the day in the



Fig 3.—Soxhlet apparatus (Messrs John B H and Croyden)

morning and to divide each feed out in separate bottles, which may then be brought to the boil by a Soxhlet or some other convenient apparatus (Fig 3)

**Measures**—Directions are given in teaspoonsful and tablespoonsful and since there is no universally accepted standard teaspoon and tablespoon this causes confusion. It is always best to measure fluids in ounces; an ordinary household tablespoon contains from  $\frac{1}{2}$  to 1 fluid oz. Glass measures or measuring jugs marked in ounces on the inside, or even a child's feeding bottle marked in ounces are much more accurate. In measuring solids, teaspoonsful vary enormously. Actually a level measure (slightly pressed down) of skimmed half cream, or full cream dried milk, weighs 1 drachm or  $\frac{1}{8}$  oz. A very heaped teaspoonful (measured in a teaspoon purchased from Woolworth's) of skimmed, half cream, or

full cream dried milk also weighs 1 drachm. A level teaspoonful slightly pressed down, is  $\frac{1}{2}$  drachm. A level tablespoonful (Woolworth's size) slightly pressed down of any of the above is equal to 2 drachm ( $\frac{1}{4}$  oz.)

**Sugar.**—This is much heavier than dried milk. One level teaspoonful equals 1 drachm. One level tablespoonful equals 4 drachms. A lump of Tate and Lyle's sugar weighs, on the average, 1 drachm. The tin measure given in Cow and Gate dried milk, if levelled off and slightly pressed down with brown sugar, weighs 2 drachms.

An English pint is 20 oz. and an American pint 16 oz. An English tablespoonful is exactly twice the size of an American tablespoonful.

### GENERAL DIRECTIONS TO MOTHERS AND NURSES

The physician should bear in mind the following four points when directing a mother or nurse who is to feed an infant artificially. These points are of cardinal importance and apply to every artificially-fed infant.

(i) **Quick Feeding**—See that the hole in the teat is a good size so that the baby can get the feed in 15 minutes easily.

(ii) **Breaking Wind**—Hold baby up for 20 minutes after each feed, until the wind is broken twice.

(iii) **Fruit Juice**—Give orange or tomato juice, rose-hip syrup or blackcurrant purée, two to three teaspoonfuls daily throughout the year, diluted with water and sweetened with sugar.

(iv) **To Prevent Rickets**—A drop of halibut liver oil, or some cod liver oil preparation, or one of the concentrated vitamin D preparations, such as Ostelin or Radio-stoleum, is required before three feeds except in the hottest summer weather. Seven hundred I.U. are required daily. (See p. 74.)

### PRACTICAL INFANT FEEDING

The theoretical quantities of food required for a healthy infant having been given in the previous pages, the student should be able to make use of the simple practical tables and formulae given below. He should bear in mind that when calculating a feed by weight, it is the *expected weight of the infant*, and not its *real weight at the moment*, on which the calculation should be based. (See p. 96.)

Providing the times of feeding are kept regularly, there is very little chance of overfeeding an infant if it is allowed to take what it will. Too often a table of amounts and weights is followed slavishly, to the detriment of the baby. In prescribing a feed sufficient should be put in the bottle to allow a little always to be left at the end of a feed, just as nature does in breast feeding. The feed for a child one pound heavier than the one being prescribed for should be given, thus leaving a little in the bottle each time, and ensuring that the infant is not underfed. Under these circumstances, of course, the infant must not be forced to take the whole of its feed when it has obviously had sufficient.

## FEEDING FROM THE FOURTH DAY TO ONE MONTH

### TABLE VI

(i) HUMANIZED DRIED MILK<sup>1</sup> FORMULA FOR A NORMAL HEALTHY INFANT FROM THE FOURTH DAY

Weight of Infant in Pounds	Measures of Humanized Dried Milk <sup>1</sup>		Water in Ounces		Number of Feeds Daily
	Per Day	Per Feed	Per Day	Per Feed	
6	12½	2	12½	2	6
7	15	2½	15	2½	6
8	17½	3½	17½	3½	6
9	20	4	20	4	5
10	22½	4½	22½	4½	5
11	25	5	25	5	5
12	27½	5½	27½	5½	5
13	30	6	30	6	5

Note—Where no measure is obtainable a very heaped large teaspoonful is approximately a drachm

(ii) FRESH COW'S MILK FORMULA FOR A NORMAL HEALTHY INFANT FROM THE FOURTH DAY TO ONE MONTH

Boiled cow's milk	½ pint (10 oz)
Water	½ pint (10 oz)
Sugar	3 level tablespoonsful

Of this mixture give the quantities shown in the table below

at 6 a.m. 9 a.m. 12 noon 3 p.m., 6 p.m. and 10 p.m. or 6 a.m. 10 a.m. 2 p.m., 6 p.m. and 10 p.m.

When baby weighs 5 lbs., give 6 feeds of 2½ oz

"	6	6	2½
"	7	6	3
"	8	5	4
"	9	5	4½
"	10	5	5
"	11	5	5½
"	12	5	6
"	13	5	6½
"	14	5	7
"	15	5	7

(1) Quick Feeding—See that the hole in the teat is a good size so that the baby can get the feed in 15 minutes easily

(2) Breaking Wind.—Hold baby up for twenty minutes after each feed until the wind is broken twice

(3) Fruit Juice.—Give orange or tomato juice rose hip syrup or black-currant purée two to three teaspoonful daily throughout the year diluted with water and sweetened with sugar

(4) To Prevent Rickets—A drop of halibut liver oil or some cod liver oil preparation or one of the concentrated vitamin D preparations, such as Osteon or Rikolatum is required before three feeds except in the hottest summer weather. Seven hundred I.U. are required (see p. 74)

(iii) UNSWEETENED CONDENSED (STERILIZED)<sup>2</sup> MILK FORMULA FOR A NORMAL HEALTHY INFANT FROM THE FOURTH DAY TO ONE MONTH

Condensed milk	5 oz
Water	15 oz
Sugar	2 level tablespoonsful

<sup>1</sup> Humanized Ambers, Albion's No. 1 Humanized Cow and Gate Humanized Glaxo Humanized T. Food

<sup>2</sup> Ideal, Libby's and Carnation Brand.

Directions Of this formula give the infant the same quantities as shown in the table for fresh cow's milk above. The same directions for quick feed or breaking with fruit juice and prevention of rickets also apply.

### FEEDING FOR A NORMAL HEALTHY INFANT FROM ONE TO SIX MONTHS

(i) **Dried milk**—It is the author's experience that after the second month the infant thrives better on a full cream dried milk than on humanized dried milk.

TABLE VIII

FORMULA FOR FEEDING ON FULL CREAM DRIED MILK FROM ONE TO SIX MONTHS<sup>1</sup>

Weight of Infant in Pounds	Full Cream Dried Milk in Drachms or Measures		Water in Ounces		Level Teaspoonful of Sugar		Number of Feeds
	Per Day	Per Feed	Per Day	Per Feed	Per Day	Per Feed	
7	8½	1½	1½	1	5	2½	6
8	10	1½	1½	2½	6	1½	6
9	12	2½	17½	3	7	1½	6
10	14	2½	20	4	8	1½	5
11	15½	3	22½	4½	9	1½	5
12	16½	3½	25	5	10	2½	5
13	17½	3½	27½	5½	11	2½	5
14	18	4	30	6	12	2½	5
15	19	4½	32½	6½	13	2½	5
16	20	4½	35	6½	14	2½	5
17	21	5	37½	7	15	3	5

Notes—When the infant reaches the weight of 15 lb. or the age of five months, the next diet should be commenced.

(ii) **Fresh cow's milk**—The formula given below is considerably stronger than the one which is used during the first month. The author thinks it is never desirable to change the strength too quickly. The competent and intelligent maternity nurse will make the change gradually from one formula to the other during the fourth week.

#### FORMULA FOR NORMAL BABY FROM ONE TO SIX MONTHS

Boiled cow's milk	1 pint (20 oz.)
Water	½ pint (10 oz.)
Sugar	3 level tablespoonsful

Of this mixture give the quantities mentioned in the table below

At 6 a.m. 9 a.m. 12 noon 3 p.m. 6 p.m. and 10 p.m. or 6 a.m.  
10 a.m. 2 p.m. 5 p.m. and 10 p.m.

When baby weighs 5 lb. give 6 feeds of ½ oz. of the above

6	6	½
7	6	¾
8	5	4
9	5	4½
10	5	5
11	5	5½
12	5	6
13	5	6½
14	5	7
15	5	7

Cow and Gate (Lancet), Doreville (Lancet), M. Hall, Ambrose, National Dried Milk, etc.

(iii) **Unsweetened Condensed Evaporated Milk<sup>1</sup>**

Condensed milk	10 oz
Water	20 oz.
Sugar	3 level tablespoonsful

Directions —Of this formula give the infant the same quantities as shown in the table for fresh cow's milk above

Quick Feeding —See that the hole in the teat is a good size so that the baby can get the feed in 15 minutes easily

Breaking Wind —Hold baby up for 20 minutes after each feed until the wind is broken twice

Fruit Juice —Give orange or tomato juice rose-hip syrup or black currant purée two to three teaspoonsful daily, diluted with water and sweetened with sugar

To Prevent Rickets —A *drop* of halibut liver oil or some cod liver oil preparation or one of the concentrated vitamin D preparations, such as Ostelin or Radiostoleum, is required before three feeds, except in the hottest summer weather. Seven hundred I U are required daily (see p. 74)

**Commencement of mixed feeding.**—When a child weighs 15 lb., that is when it is aged about five months, it receives about 20-25 oz. of cow's milk in the day. This amount ought to be the maximum which any infant receives, and from this time on the amount should tend to decrease rather than increase. As the child requires more food therefore it should be added in the form of starches, since the full amount of carbohydrate as sugar, namely two ounces, has already been added. The feeds are four hourly, and it will be found useful to feed as shown below

This régime is arrived at by slow stages, each addition taking two to three days, and half amounts only being given until the child adjusts itself to the additions. For children who find difficulty in managing the cereal, a preliminary week on Benger's, or Savory and Moore's, at 10 a.m. and 6 p.m. is a good intermediate stage. Some infants prefer the cereal as a semi solid from a cup, well sweetened, with a little milk added. It is undoubtedly an advantage to push on with spoon feeding at the earliest possible moment. When the mixed feeding is attempted as an addition to breast milk, the administration of the food by spoon rather than by bottle is much more convenient. It is often an advantage to add some cow's milk to the soup, together with one of the homogenized vegetable preparations

Probably it is best to start by making the addition to the 10 a.m. feed, then to the 6 p.m. feed, and finally to the 2 p.m. feed. The greater the variety of cereals the more likely is success to be achieved. Wheat starch such as in rusks and Robb's biscuits, is less rachitic than oat or barley starch, rice flour being intermediate. Oat flour should certainly not be given at more than one feed in the day.

**DIET FOR A NORMAL HEALTHY INFANT FROM SIX TO NINE MONTHS OLD**

		Weight 15 to 18 lb			
Feeding Times		6 a.m.	10 a.m., 2 p.m.	6 p.m., 10 p.m.	
6 a.m.	Milk	5 ounces	} or {	National Dried Milk	7 measures
	Water	2 ounces		Water	7 ounces.
	Sugar	1 heaped teaspoonful		Sugar	1 heaped teaspoonful

<sup>1</sup> Ideal, Libby's and Crumion Brands

**10 a.m.** Mixture as above, to which has been added one to three heaped teaspoonsful of any of the following —Chapman's Mature Wheat Food, Sister Laura's Food, Gruel & Cream of Rice, Robinson's Patent Groats or Patent Barley, Farax No 1 or Nerve Food. (See note on cooking) Half a teaspoonful of the yolk of a lightly boiled egg should be slowly introduced along with this feed and gradually increased to two teaspoonsful if well tolerated

**2 p.m.** Milk 5 ounces  
 Sugar 1 heaped teaspoonful  
 Bone and vegetable broth 2 tablespoonsful (see note below)

} or {

National Dried Milk 5 measures  
 Water 5 ounces  
 Sugar 1 heaped teaspoonful  
 Bone and vegetable broth 2 tablespoonsful (see note below)

(Heinz's Baby's Brand or Nestlé's homogenized vegetables one to two tablespoonsful can be given daily in addition to the broth)

**6 p.m.** The mixture as at 10 a.m. but with one to three teaspoonsful of a different cereal (No egg to be given at this feed.)

**10 p.m.** Exactly as at 6 a.m.

**Fruit Juice**—Orange or tomato juice two to three teaspoonsful, diluted with water and sweetened with sugar or a similar quantity of black currant puree or rose-hip syrup diluted with water should be given daily. Should none of these be obtainable the alternative is 50 mgm. of ascorbic acid. A convenient time for this is between 8 and 10 a.m. or at tea time.

**To Prevent Rickets**—One half teaspoonful of cod liver oil or one drop of halibut liver oil should be given immediately before three feeds daily except in the very hot summer weather.

**Note on Cooking**—All the food should be brought to the boil. The cereal for the 10 a.m. and 6 p.m. feeds, such as groats or cream of rice needs to be cooked for at least 10 minutes, or from half an hour to an hour if cooked in a double saucepan. It may be added to the rest of the feed and the whole cooked for the specified time or it may be cooked with water and then stirred into the rest of the feed when it is thoroughly cooked.

**A B**—If this thick feed is given from a bottle it is necessary to make a large hole in the teat. It is better to spoon feed from a cup.

**Bone and Vegetable Broth**—Take 1 lb. of veal or beef bones or calves' feet, well broken up. (During war time chicken or rabbit bones may be substituted or where bones are not available a little meat such as beef, veal or lamb cut into small pieces.) Cover with water and add one teaspoonful of vinegar. Occasionally, say once a fortnight add a small piece of calves' or ox liver (about 2 oz.). Simmer for from four to seven hours. Now add vegetables (carrots, cauliflower, swedes, parsnips, green vegetables and potatoes). Simmer for one more hour strain and allow to set. The broth is best cooked in a double saucepan and should keep for three days in a cool place.

**A B**—Broth like this can be purchased ready made from Bicknape's Ltd., Nursery Food Specialists Dept 12 Welwyn Garden City, Herts. (Telephone, Welwyn Garden 428)

### DIET FOR A NORMAL HEALTHY CHILD FROM NINE MONTHS TO ONE YEAR OLD

**A B.**—The transition from the previous diet to this one should be slow, taking about two weeks.

**On waking** The juice of an orange or tomato diluted with water and sweetened with one teaspoonful of sugar. Alternatively, one of the following —  
 Bottled orange or lemon squash—1 tablespoonful  
 Black currant puree—2 to 3 teaspoonful,  
 Black currant juice—2 to 3 teaspoonful  
 Juice from tinned tomatoes—1 tablespoonful  
 Rose hip syrup—2 to 3 teaspoonful,  
 Government fruit juice—2 to 3 teaspoonful

(Note—These amounts are approximate and should be adjusted to suit the individual child) If none of the above is available 50 mgm. of ascorbic acid (vitamin C) should be given. Milk should never be given at this time.

- Breakfast**  
8 a. m.
- 1 Half a cup of cereal such as Chapman's Entire Wheat Food, Barley Farex, Cream of Wheat, Wheatena, Groat's M O F or rusks (see directions below for making these) and milk. Robb's biscuits with milk fed with a spoon from a cup.
  - 2 Four mornings in the week give half an egg and two mornings toast crumbs or crisp toast fried in lard or chicken dripping. One morning a little steamed fish can be given.
  - 3 Eight ounces of milk (including that given with the cereal) (Vary the first course as much as possible).
- Dinner**  
12 30 p. m.
- 1 One to two heaped tablespoonsful of mashed potato (potatoes should be cooked in their skins to retain the vitamin C) and one to two heaped tablespoonsful of sieved vegetables (carrot, spinach, swedes, brussels sprouts, etc.) moistened with four tablespoonsful of broth (see below). Heinz Libby's Nestlé's or Brand's homogenized vegetables will be found most suitable at this age. As the child approaches one year of age the following may be added: pounded chicken, white fish, sweetbread, brains or underdone scraped steak, or very finely minced meat or rabbit.
  - 2 One to two heaped tablespoonsful of ground rice tapioca, sago, semolina or egg custard pudding with sugar and jelly, apple sauce or homogenized prunes (Brand's).
  - 3 Four ounces of milk at this meal (which should be used for making up the pudding).
  - 4 Water to drink.
- Tea**  
4 30 p. m. to 5 p. m.
- 1 Rusks and milk or Robb's biscuits and milk or (preferably) rusks spread with butter or thin brown or white bread and butter.
  - 2 Junket or stewed fruit or custard.
  - 3 Eight ounces of milk.
- 10 p. m.
- Four ounces of milk if necessary, but the child should not be awakened for it. Normally, the 10 p. m. bottle should be completely discontinued by the 9th month.

Milk—All milk given should be boiled.

To prevent rickets and ensure good teeth—One of the following should be given: one teaspoonful of cod liver oil emulsion, or one third of a teaspoonful of Government vitaminized oil, or one teaspoonful of cod or halibut liver oil with malt or two drops of halibut liver oil three times daily, throughout the year except in the very hottest summer weather.

Bone and vegetable broth—See p. 44.

Rusks—Cut slices of a stale loaf of brown or national bread about half an inch thick cut into fingers dip into a milk and water mixture, sweetened with sugar and place in a slow oven to crisp.

## DIET FOR A NORMAL HEALTHY CHILD FROM ONE TO TWO YEARS

On waking The juice of an orange or tomato diluted with water and sweetened with one teaspoonful of sugar. Alternatively one of the following—

- Bottled orange or lemon squash—1 tablespoonful,
- Black currant purée—2 to 3 teaspoonsful,
- Black currant juice—2 to 3 teaspoonsful,
- Juice from tinned tomatoes—1 tablespoonful
- Rose hip syrup—2 to 3 teaspoonsful
- Government fruit juice—2 to 3 teaspoonsful

(Note—These amounts are approximate and should be adjusted to suit the individual child) If none of the above is available 50 mgm. of ascorbic acid (vitamin C) should be given. Milk should never be given at this time but one small rusk may be offered (See above).

- Breakfast**  
8 a m
- 1 One tablespoonful of any of the following —Well cooked groats, Cream of Wheat or Wheatena, rusks in milk, or Robb's biscuits in milk (In warm weather stewed fruit and crisp toast may be given, but see that the child's appetite is not satisfied with this course)
  - 2 Toast crumbs fried in bacon fat, or a small rasher of crisp bacon or half a soft boiled egg with breadcrumbs (four days in the week) Lightly cooked dried egg may be used during war time or pounded plaice or sole or fresh herrings
  - 3 Eight ounces of milk (including that used with the cereal)
- Dinner**  
12 30 p m
- 1 One level tablespoonful of any of the following —Fish (boiled or steamed) or pounded chicken brains, kidney or rabbit, or sweet breads, or scraped raw or underdone steak, or Irish stew or lightly cooked liver
  - 2 One heaped tablespoonful of boiled, baked or mashed potato (potatoes should always be cooked in their skins), and one heaped tablespoonful of one of the following —sieved sprouts, cabbage spinach, greens, cauliflower, carrot, swedes, parsnips or lettuce (These are all best steamed)
  - 3 Milk pudding (Groult's cream of rice, tapioca, etc.) with stewed apples, prunes, junket or custard
- Tea supper**  
4 30 to 5 p m
- 1 Rusks or pulled bread (Zwieback), or crisp toast, or thin bread and butter, with a little honey, seedless jam, jelly, custard, junket or stewed fruit and a small piece of sponge cake
  - 2 Between one year and eighteen months Robb's biscuits or rusks may be given soaked in warm milk
  - 3 Eight ounces of milk (including any used with biscuits)
- 6 15 p m
- It ought not to be necessary to give anything after tea supper. If the child eats a poor tea, however, some of the milk and rusks may be kept and offered before he is put to bed, but this should not be done as a routine

**Milk.**—One pint of milk per day should be sufficient, including that used in cooking  
*All milk should be boiled*

**Sweets.**—Plain boiled sweets, such as barley sugar or fruit drops, may be offered after dinner and tea the teeth to be cleaned immediately afterwards

To prevent rickets and ensure good teeth—One of the following should be given One teaspoonful of cod liver oil emulsion, or one third of a teaspoonful of Government vitaminized oil, or one teaspoonful of cod liver oil or halibut oil with malt, or two drops of halibut liver oil —three times daily, throughout the year, except in the very hottest summer weather

**Note.**—Some of the solid constituents in this diet must be introduced slowly during the period from one to two years, the whole diet being unsuitable at first. Red meat should be introduced slowly and in very small quantities.

## DIET FOR A NORMAL HEALTHY CHILD FROM TWO TO SEVEN YEARS

- On waking**
- The juice of an orange or tomato diluted with water and sweetened with one teaspoonful of sugar. Alternatively one of the following —
- Bottled orange or lemon squash—1 tablespoonful,
  - Black currant puree—2 to 3 teaspoonsful,
  - Black-currant juice—2 to 3 teaspoonsful,
  - Juice from tinned tomatoes—1 tablespoonful,
  - Rose hip syrup—2 to 3 teaspoonsful,
  - Government fruit juice—2 to 3 teaspoonsful.

(Note —These amounts are approximate and should be adjusted to suit the individual child) Milk should never be given at this time, but one small rusk may be offered (See p 45)



## DIET FOR 2-7 YEARS

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- |                                   |  |
|-----------------------------------|--|
| Breakfast<br>8 a.m.               | <ol style="list-style-type: none"> <li>1 Porridge, cornflakes, Farax, Grape nuts shredded wheat, Cream of Wheat, Wheatena, Wheatalax puffed rice or some dried cereal, with milk, in place of one of these, in warm weather, stewed apples, prunes, plums cherries, figs, etc (pureed for younger children) Do not satisfy the child's appetite with this course</li> <li>2 An egg or dried egg (three mornings per week) or —</li> <li>3 Tomatoes and a rasher of crisply fried bacon (three mornings), or —</li> <li>4 Fish or lightly cooked liver or kidney (one morning)</li> <li>5 Crisp toast or rusk spread with butter</li> <li>6 Eight ounces of milk (including that given with the cereal)</li> </ol>  |
| Dinner<br>12 30 p.m.              | <ol style="list-style-type: none"> <li>1 Two tablespoonful of any of the following: cutlet mince or fish stew underdone beef, steak (finely cut up), sweetbreads sheep's heart tripe, liver kidney, brains, fish fish pie, chicken or rabbit (The meat should be minced for young children)</li> <li>2 One to two heaped tablespoonful of boiled mashed or baked potato (All potatoes are best cooked in their skins to preserve the vitamin C)</li> <li>3 One to two tablespoonful of mashed carrot cauliflower sprouts peas, beans spinach, swedes parsnip greens, cabbage etc (These should be pureed for the younger children) All raw vegetables should be either steamed or cooked in very little water which should afterwards be added to soups and gravies</li> <li>4 Milk pudding stewed fruit, steamed pudding, custard or junket or blancmange—one to two heaped tablespoonful Water to drink</li> </ol> |
| Tea Supper<br>4 30 to<br>5 0 p.m. | <ol style="list-style-type: none"> <li>1 Crisply toasted wholemeal or brown bread thin bread and butter, rusks and butter or cream cheese, mustard and cress water cress, tomato, marmite, honey, jelly, blackcurrant purée or seedless jam sandwiches</li> <li>2 Stewed fruit or jelly with the bread and butter</li> <li>3 A small piece of sponge cake</li> <li>4 Eight ounces of milk</li> </ol>   |
| 6 30 p.m.                         | <p>If the child has left some of his tea, the remnants may be offered at 6 30 p.m. but as a rule it will be found best not to give anything more before bed</p>  |

Milk—One pint of milk per day should be sufficient, including that used in cooking. Some children prefer milk flavoured with cocoa, coffee essence or tea. All milk must be boiled, or pasteurized.

Fruit—A child will not lack Vitamin C if the fruit juice is given and also ripe fresh fruit whenever available. (These should be pureed for the younger child, and soft fruits only used). Additional watercress, mustard and cress, brussels sprouts and potatoes cooked in their jackets, together with one 50 mgm. tablet of ascorbic acid daily will provide vitamin C for those not receiving the Government fruit juice.

Sweets—Plain boiled sweets, such as barley sugar (barley malts or barley brights) or fruit drops, or an occasional toffee may be offered after dinner or tea (the teeth to be cleaned immediately after).

To prevent rickets and ensure good teeth—One teaspoonful of cod liver oil emulsion, or one third of a teaspoonful of Government vitaminized oil, or one teaspoonful of cod liver oil and halibut liver oil and malt, or two drops of halibut liver oil, should be given three times daily throughout the year, except in the very hottest summer weather.

## DIET FOR CHILDREN OF SCHOOL AGE

The growing child requires a substantial and mixed diet which should contain fresh meat, an abundance of fresh vegetables and fruit, butter, milk and occasionally eggs, and should be properly balanced with sugar and starch. A child of 12 years requires as much food as either parent.

Fruit—Either on waking or to commence breakfast, fresh fruit is most advisable. A ripe apple, orange, grapes, grapefruit or bottled or tinned fruit or tomato juice or squash, such as Via-ora, should be given.

- Breakfast**
- 1 Porridge, Grapenuts, shredded wheat, Force, puffed rice, Wheatena, Cream of Wheat, groats, creamed barley, etc
  - 2 An egg, soft-boiled, scrambled or fried, with or without bacon, three or four mornings in the week. On the other mornings, crisply done thin rashers of bacon, with tomatoes or fish (sole, plaice, herring or kipper). Well browned, mashed potatoes occasionally with the main dish
  - 3 Crisp thin toast, butter and marmalade
  - 4 A glass of milk or weak tea and milk
- Dinner**
- 1 Cutlet, fresh mince or stew, a cut of roast beef, steak or mutton (four days in the week) and chicken, fish, sweetbreads, liver, kidney, rabbit, brains on three days
  - 2 Boiled, baked or mashed potato (which should always be cooked in their skins) with carrots, cauliflower, parsnips, swedes, or green vegetables such as spinach, sprouts, cabbage, or peas or beans. Green salads
  - 3 Stewed fruit and milk puddings or custard, or sponge or steamed puddings. *Water to drink*
- Tea Supper** (See note below when tea and supper are given separately)
- 1 Thin bread and butter, with cooked fruit, such as baked apple or stewed figs, prunes, rhubarb, green gages, cherries, etc, or milk pudding or honey jam cream cheese, lettuce, Marmite, water cress tomato or peanut butter sandwiches
  - 2 Sponge cakes or scones. *Milk or weak tea or cocoa, with milk*

*Note*—As the child approaches the age of 7 or 8, a more substantial tea supper is required. Occasionally an egg may be introduced or vegetable broth, or fish, or macaroni or spaghetti, or kedgeree, or beans and tomatoes, fried with bread crumbs, or cheese, or a milk pudding may be added to the meal. At the age of about 9 or 10 however, it is best to give a very light tea at 4.30 p.m. consisting of one cup of milk or weak tea with milk, and one small biscuit. Supper should be given separately at 6 or 6.30 p.m.

**Suitable suppers**—Kidney on toast with rice pudding. Ham with salad and blanc mange. Fish and potato with baked apple or prunes. Sausage and potato with fruit or jam trifle. Baked eggs done with tomatoes, and milk jelly or baked egg custard. Macaroni and cheese or cauliflower cheese and caramel pudding. Thick vegetable soup and coffee blanc mange or ice cream. Sardines on toast with stewed fruit and custard. Sweetbreads or brains with stewed fruit and cream. Minced chicken or rabbit on toast with white sauce and junket.

**Milk**—Each child requires one pint of milk per day. In exceptional cases, where it is well tolerated, more may be given with advantage.

**Sweets**—Plain boiled sweets such as Diamanta (barley sugar, barley malts or barley brights or fruit drops) or Macintosh & toffee, may be offered after dinner or tea (the teeth to be cleaned immediately afterwards).

## CHAPTER IV

### THE CARE AND FEEDING OF THE PREMATURE INFANT

**Definition of prematurity.**—In conformity with the standard in international use an infant whose birth weight is  $5\frac{1}{2}$  lb (approximately 2 500 grammes) or less, shall be taken for purposes of comparison of records as either immature or prematurely born, regardless of the estimated period of gestation

**TABLE IX**  
**DEATHS DUE TO PREMATURETY**  
(England and Wales)

Year	No of Live Births	No of deaths of infants under 1 Year certified as prematurity	No of deaths of infants under 4 Weeks certified as prematurity	No of deaths from all causes under 4 Weeks
1940	607,099	8,598	8 074	17 503
1941	537,223	8 477	7 883	16 746
1942	654 030	8 861	8,308	17 676

The above table shows that —

- (a) Of the total number of deaths certified as due to prematurity in infants under one year the percentage occurring in the first four weeks of life was as follows —

1940	93.91 per cent
1941	92.76 per cent
1942	93.76 per cent

- (b) Of the total number of deaths occurring in the first four weeks of life the percentage certified as due to prematurity was as follows —

1940	46.13 per cent
1941	47.07 per cent
1942	47.00 per cent

**Causes of prematurity**—It is necessary to consider, first why the child has been born prematurely, and the possible effects on it of these pathological factors and secondly, what damage may have occurred during or after birth. Some of the factors which, acting through the mother, may produce premature labour, are syphilis, nephritis (toxæmia of pregnancy and hydramnios), anaemia and general poor health during pregnancy due to an insufficient and badly balanced diet (see p. 25) heart disease, acute infectious fevers, chronic infections of the uterus, multiple pregnancy (twins or triplets), poisoning of the mother by lead, arsenic or other metals. Malformations or abnormalities in the foetus itself, such as hydrocephalus or absence of the abdominal wall, may cause premature birth. Miscarriage occurs sometimes without any known cause.

TABLE X

*Probable Causes of Prematurity<sup>1</sup>*

<i>Maternal Condition</i>	<i>No of Cases</i>
Multiple pregnancy	15
Maternal toxæmia	13
Rheumatic heart disease	2
Repeated history of prematurity or miscarriage	4
Cæsarean section	2
Acute infection of the mother —	
Pyelitis	1
Pyelo nephritis	1
History of anti syphilitic treatment	1
	<hr/> 39 <hr/>

**Care of the premature infant.**—In the management of the infant the chief factors are the preservation of its body heat, that is, the prevention of a subnormal temperature and careful administration of sufficient and suitable food.

**Methods for the maintenance of body-temperature**—The premature infant has no means within itself of keeping its temperature up to normal and regulating its heat loss. Heat must therefore be applied from outside. Its breathing is so shallow its circulatory system so inactive and its metabolism so slowed that it is not able to manufacture sufficient heat for itself unless that heat be well conserved. One of the chief problems is to keep the temperature of these infants within normal limits.

The baby should be received at once into a jacket of cotton wool, arms and legs being done up in this material, and the buttocks being kept free, with a separate pad for a napkin. The infant is then wrapped round in a shawl and deposited in a basket or bassinette, for which some method of heating has been devised. A simple method for poor parents is to hang several stone ginger beer bottles filled with hot water just within the basket. Hot water bottles placed one at each side and one at the foot of the basket, and changed frequently, should suffice to keep the baby's temperature up to normal. A powerful electric light or an electrically heated pad placed over the infant is effective, and a great convenience, as the heat can be regulated. A thermometer should be placed inside the coverings, and inspected each hour. At the same time the infant's temperature should be taken, and the amount of applied heat required to maintain it will thus rapidly be ascertained.

The question of incubator rooms, or hospital treatment, often arises. As a rule an infant who is skilfully nursed is safer at home than anywhere else. Often, however, this is not financially practicable. If possible, the child should be kept in a specially heated room at 70–80°, with some additional moisture in the air, it then only needs a cot with an electric pad placed over the infant (not under).

No attempt should be made to bath a premature infant during the first few weeks of life. It should be kept well rubbed with olive oil and the buttocks sponged from time to time. Weighing should be done in its clothing, and when an opportunity arises and the clothing is changed the

<sup>1</sup> J. C. Epe and P. J. W. Miller. Report of an Investigation into the Cause of Infant Mortality in Newcastle-upon-Tyne during the year 1909.

weight of this can be deducted, so that the weight of the actual child is then ascertained. When a weight of 5 to 6 lb is attained normal clothing can be used.

**Prevention of infection.**—The premature infant is particularly liable to infections with the most disastrous consequences. Too great precautions cannot possibly be taken. The nurses must wear masks which should be frequently changed, and be of the most efficient type. Scrupulous cleanliness over bottles, teats and all utensils is necessary. Doctors and students should be excluded from examining an infant unless there is some real reason, and all relatives kept away for the time being. A slight naso-pharyngeal catarrh is quite sufficient to upset weeks of work and precipitate a fatal diarrhoea or pneumonia.

**The feeding of the premature infant.**—The caloric requirements of the premature infant are vastly higher in proportion to its body weight than those of a normal infant. The problem is therefore one of securing a food sufficiently high in caloric value to make it gain and sufficiently digestible to allow the intestinal tract to cope with it in large enough quantities to promote growth. There is a very fine line between over-feeding and keeping the intestinal tract healthy and underfeeding but supplying sufficient calories. The slightest swing to either side proves equally disastrous. It is difficult to say whether more premature infants are lost from underfeeding or from overfeeding—probably from the former as it is not commonly known that such infants require relatively enormous quantities of food.

**Breast-feeding.**—Obviously, the great digestibility of breast milk makes it ideal for these babies, since it can be dealt with in relatively large quantities without fear of digestive upset in a way with which no other food can compare.

There is no doubt that where the mother can breast feed her infant the chances of its survival are greatly increased. Very often, however, if the infant be very premature, in which case suckling is all the more necessary, the mother is quite unable to supply breast milk, as it has not appeared. This is especially true with the first baby. Breast milk can now (1944) be purchased in any quantity from Queen Charlotte's Hospital, Marylebone Road, London, W1. Friends or neighbours will occasionally part with some of their breast milk for the time being and give the infant a start. Should the mother be able to breast feed the child, it should be put to the breast twice in the second 12 hours and thereafter at three-hourly intervals for a gradually lengthening period commencing with three minutes. If the infant is too feeble and undersized to suck properly, the breast milk may be expressed and given as described on p. 53.

A mixture of equal parts of breast milk and water should be given to the infant by pipette or Brel feeder, or directly into the stomach with a fine urethral catheter, size 5 or larger. In the latter case it should be run in very slowly, carefully pinching the tube when it is being withdrawn, so that the milk does not get into the trachea.

**Times of feeding.**—Certainly for the first fortnight the premature infant on the breast will, if under 3½ lb, require two-hourly feeding by day and three hourly feeding by night, if over 3½ lb three hourly feeding by day.

and four hourly feeding by night. For times of feeding see p 53. More frequent feeds are required where feeding is artificial.

*Amount of breast milk required*—A normal healthy infant requires about  $2\frac{1}{2}$  oz per pound of its body weight per day, that is, an infant weighing 6 lb at birth very quickly obtains about 15 oz of breast milk in the day from its mother ( $2\frac{1}{2} \times 6$ ). The premature infant requires relatively a greater amount in the day for each of its pounds and fails to thrive on anything less than about 3 oz per pound body-weight per day, that is, a premature baby weighing 4 lb will require about 12 oz of breast milk in the day before it begins to gain weight properly. Waiting for the milk to appear, i.e., until the third or fourth day, is more trying for the premature infant than for the normal infant. In the second 24 hours the infant requires  $1\frac{1}{2}$  oz for each of its pounds in the day, whereas by the fourth 24 hours this has risen to 2 oz per pound body weight per day. By a fortnight at latest the full amount of 3 oz per pound body weight per day should have been reached.

From the second day additional breast milk and water, equal parts should be given after the baby has been put to the breast, until a good flow from the breast begins.

*Artificial feeding. Choice of food*—If breast milk is not obtainable then some artificial food must be used. The premature baby has not the same ability as the full term child to manage the various food elements. Sugar is metabolized efficiently, and relatively high carbohydrate feeds are recommended. Protein or curd however, must be very carefully modified as there is no doubt that the infant is most susceptible to curd indigestion in the first few weeks of life. Fat seems to be managed fairly well.

The following foods are suggested, in this order —

- 1 *Humanized dried milk*—Eralac, Humanized Cow and Gate, Humanized Trufood, Humanized Glaxo, Osterimilk No 1. These are best made up one measure to the ounce of water, in which case breast milk is reconstituted. For the first week, however, a weaker mixture could be offered.
- 2 *Unsweetened evaporated condensed milk*—This is best made up with 1 oz of condensed milk to 3 oz of water, and one rounded teaspoonful of sugar.
- 3 *Lactic acid milk* (see Appendix, p 102)—This should be made up with an equal quantity of water, and one rounded teaspoonful of sugar, to each 3 oz of the mixture.
- 4 *Fully peptonized milk*, with water and sugar, as above.

The child should gradually be given an amount equal to 3 oz of any of these mixtures for each of its pounds in the day. This amount should be reached by about the 14th day. As an example, a 4 lb premature baby requires 12 oz of the mixture in the day. The child is to receive 10 feeds, starting with one ounce at each feed, the amount can be increased by adding an extra teaspoonful to each feed until the whole 12 oz is given daily. Daily weighing is most necessary. Should the infant show a steady

gain no increase in the feed should be given but if the weight is stationary then the feed should be cautiously increased

*Times of feeding*—The infant under 8½ lb should be fed at 6 8 10 12 2 4, 6, 9, 12, and 3. When it is thriving well these periods should gradually be lengthened until the infant is fed three hourly by day and four hourly by night

For example an infant is born one month prematurely weighing 3 lb. It should be fed at 6 8 10 12 2 4 6, 9, 12 and 3 and be given ultimately 9 oz of breast milk in the day, or 9 oz of the artificial mixture. Supposing the "Ideal" milk mixture is the most suitable commence by giving half an ounce in a bottle with an easy hole in the teat at each feed. The child should be weighed each day, and each day the bottle should be increased by one teaspoonful, that is having got the child accustomed to half an ounce by the fourth or fifth day, 5 teaspoonsful should be given and if the child is not gaining, increased to 6 then to 7 and finally to 8 teaspoonsful. As soon as the infant begins to put on weight an ounce per day or more no further increase in the feed will be made but at any sign of stationary weight the feed should be increased by a further teaspoonful.

*Difficulties in administering the feeds.*—In some infants there is obstinate anorexia. The baby sleeps continuously and shows no desire for food. The mouth should be examined carefully to make sure it is not sore. It may be necessary for a period of 24 hours to give half normal saline or boiled water sweetened with sugar.

Should the infant be extremely weakly and unable to suck the breast or bottle properly, the Breck Feeder, or what is known as the Premature Tube Feeder, sold by Messrs John Bell and Croyden Wigmore Street London, W 1, will be found a great help. This can be improvised by placing on the end of a glass tube a small teat made from the rubber end of a fountain pen filler, in which a very fine hole has been pierced. On the other end of the glass tube an unperforated teat or finger stall is placed, and the tube filled with milk. Gentle pressure on the unperforated end forces the milk to flow from the opposite end of the tube, and the baby gets its food with little or no work. Glaxo and other firms place on the market for premature babies a bottle which is supplied with extremely small teats and is most useful. Should the infant refuse all food for any length of time it may be given its food by nasal or gastric tube. This is very seldom necessary.

*Drugs*—Moncreiff<sup>1</sup> recommends small doses of thyroid extract  $\frac{1}{16}$  grain per pound body weight per day for premature infants and considers that the metabolism is stimulated and that they are definitely benefitted. Vitamins in the form of halibut liver oil, one drop three times daily, should be given from the second week onwards. Marmite or Ryzamin B should be added to one feed in the day, and vitamin C in some form offered, certainly by the second week.

It is the author's custom to alternate some iron preparation with halibut liver oil, a week of each in rotation, and he suggests that one grain of ferrous sulphate, in a suitable solution should be added to three feeds in the 24 hours, from the third week onward, alternating with the halibut oil.

**Sequelæ and prognosis** <sup>1</sup>—*Height and weight* Although there is no absolute uniformity of opinion the majority of premature infants seem to attain average height but a very large proportion remain somewhat under weight up to puberty There is some evidence that the smaller the birth weight the greater is the likelihood of low weight during childhood

*Mental development*—There is no uniformity of opinion but broadly speaking it may be said that premature birth unassociated with intra cranial injury does not affect the mental development and that neither the period of gestation nor the weight at birth has any bearing on the ultimate general intelligence Disease and ill health in the mother before the birth do not apparently affect the general intelligence

*Cerebral hæmorrhage*—From 15 to 30% of surviving premature infants show evidence of cerebral damage and cerebral abnormality with subsequent mental defect whereas in full born infants the incidence is about 6%

*Mortality*—Of those premature infants dying in the first month of life two thirds fail to survive the first 48 hours All things being equal the older the premature infant the better its chances of life and a decrease in death rate accompanies an increase in birth weight On the average 50% of the neonatal mortality occurs among premature infants

*Tendency to anæmia and rickets*—In the last month of intra uterine life the infant is largely supplied with calcium and iron from its mother If born prematurely it has failed to benefit in this way with consequent incipient anæmia and rickets To combat the rickets habit use oil as described above should be instituted early and once the infant has started to thrive real or artificial sunlight baths should be given Once the digestion has improved say at the age of 3 to 4 months stronger milk mixtures should be given if artificially fed and mixed feeding should begin by the fourth or fifth month at the latest With good care the teeth of premature infants need show no higher incidence of dental caries than those of normal children

The anæmia<sup>2</sup> should be treated by the administration of iron Trans fusions are seldom necessary but should be given where the anæmia is marked Erythroblastosis should be excluded (see p 68) Iron seems to help once the anæmia has reached its maximum about the fourth week For prescriptions see p 178

**"Blue turns" in premature infants**—Premature infants are extremely liable to sudden attacks of cyanosis or blue turns There may be a variety of causes for this The infant may have had a slight hæmorrhage round about the respiratory centre in the medulla Or the trouble may be due to a congenital atelectasia of the lungs, a portion of one or both having failed to expand The child may have congenital

<sup>1</sup> R. B. Illingworth *Arch. Dis. Child.* The Mortality of Premature Infants, No 74 1932 xiv 15  
J. Hoss, C. J. M. Br and P. F. Barthelemy "The Physical and Mental Growth of Prematurely Born Children," University of Chicago Press, 1934  
A. Toy *Advances in Pediatrics*, London, 1931 Heinemann vol. 1 p 191

<sup>2</sup> I. C. Parsons and I. C. Hawkesley, *Anæmia in Childhood*, Part III (The anæmiatopoietic anæmia) (Deficiency diseases of the erythron) Nutritional Anæmia and the Anæmia of Prematurity Scurvy and Celiac Disease *Arch. Dis. Child.* 1933 viii 133  
b. van Creveld and W. T. Heybroek *Rev. française de F. d. de Paris* 1931 viii 135



heart disease, that is, owing to its prematurity there may be a communication between the two sides of the heart or a patent ductus arteriosus. Finally, attacks of sudden cyanosis occur in infants who become unduly dehydrated. This is seen occasionally where an infant has refused its food or has slept so soundly that it could not be awakened for its feeds. Insufficient fluid is taken and on possibly the second, third or fourth day a sudden attack of cyanosis with difficulty in breathing occurs. Fluids rapidly cure it. Various observers, however, have quoted cases in which blue turns proved fatal and no explanation can be offered.<sup>1</sup> When these attacks of cyanosis come on, the infant is best kept quiet, fed in its cot and not picked up or handled in any way. Oxygen containing 5 per cent  $\text{CO}_2$  should be administered or the infant should be placed in an oxygen tent.

<sup>1</sup> G. F. Still. Attacks of Arrested Respiration in the New born, *Lancet* 1923 i 431.

## CHAPTER V

### DISEASES AND INJURIES OF THE NEW-BORN

ALTHOUGH the infant mortality during the first year of life has fallen very steeply from more than one hundred to fifty seven deaths per thou and live births the neo natal death rate has fallen very little during the same period. The neo natal period is the first four weeks of life and the death rate has fallen in the last 37 years from 41 to 26 per thousand. The causes of this mortality and their prevention are discussed in this chapter.

TABLE VI  
BIRTH AND MORTALITY RATES FOR THE YEARS 1900 TO 1942

Year	Birth rate	Neo natal mortality rate	Infant mortality rate
1900	27.3	41	199
1910	25.1	38	105
1920	25.0	30	80
1930	16.3	31	60
1937	14.9	30	57
1940	15.0	28	50
1941	14.4	27	60
1942	15.6	26	49

It has generally been supposed that this high neo natal death rate was due to congenital malformations incompatible with life. Cruickshank who has written a Medical Research Council Report on child life investigation under the title of Causes of Neo natal Death suggests that congenital malformations form only a small proportion of the true causes of this high mortality.

It is not generally realized that prematurity is the direct cause of approximately one half of the deaths in the neo natal period. (See p. 49). Spence and Miller in their investigation<sup>1</sup> of infantile mortality in Newcastle 1930 found that out of 198 neo natal deaths 15 were due to congenital malformations 38 were born dead 17 were premature there was one respiratory infection one alimentary infection 6 unknown infections 3 skin infections 1 case of whooping cough 1 blood condition 3 accidents and in 19 the cause of death was unknown.

**Examination.**—The student in examining a newborn infant should notice particularly whether the infant cries breathes and sucks normally and whether there is any tendency to twitching or convulsions. He should examine the exterior of the body for bruising and the bones for fractures. He should note whether the limbs move normally examine the heart for congenital abnormalities and should take particular note of the umbilicus.

<sup>1</sup> From the Annual Reports of the Chief Medical Officer of the Ministry of Health.

<sup>2</sup> J. O. Spence and F. J. W. Miller Report of an Investigation into the Causes of Infantile Mortality in Newcastle-upon-Tyne during the year 1930.

<sup>3</sup> Graham, *Arch. Dis. Child.*, 1935, x, 310.

**Loss of weight.**—As a rule an infant loses an ounce or more per day during the first four days. By the tenth day, however, it should have regained this loss, and may be even one or two ounces above its birth weight. From this time on a gain of one ounce a day for the first 100 days is the rule. Much of this loss can be prevented by giving drinks of sugar water until the breast milk comes in. When the milk appears early and is abundant, there may be little or no initial loss of weight.

**Dehydration fever.**—Frequently the colostrum is small in quantity, and late in appearing, and the infant is actually starved of fluid in the first few days of life. The urine is scanty, and the temperature tends to rise to 100 or 101° F. On the appearance of the breast milk the temperature tends to subside and the infant to thrive. It appears then that the temperature is produced by the infant's reaction to a shortage of fluid and dehydration can be prevented by seeing that the infant gets an adequate amount of fluid in the first three or four days after birth.

In the first 24 hours the infant should be given 4 oz. of fluid (5 per cent sugar water is suitable). In the second 24 hours the amount should be 6 oz. on the third day, 8 oz. and by the fourth day not less than 10 oz., on the average. These figures of course include whatever breast milk the mother is able to give, the amount of which can be ascertained by test feeds. (See pp 28 and 74)

#### JAUNDICE OF THE NEW BORN (ICTERUS NEONATORUM)

Almost every infant shows some degree of jaundice shortly after birth but in at least 80 per cent this is well marked. It appears on the second to fifth day, and lasts for about a week. It causes few or no symptoms and requires no treatment. There is no anemia, cedema, hæmorrhage, enlargement of the liver or spleen, or mortality. This is a physiological hæmolytic which is apparently due to a rise in the oxygen tension of the blood accompanying the change-over from placental to pulmonary respiration.

While *in utero* the infant has been in a relatively rarified atmosphere and a polycythæmia has resulted, the red count sometimes reaching 6 or 7 million. After birth these redundant red cells are hæmolyzed with resulting jaundice. There is a positive indirect van den Berg reaction in the serum and, when measured quantitatively by the icterus index, it gives values of about 10 to 20 units. When the index is about 12 to 15 units the jaundice is plainly visible in the skin. There may be a small number of nucleated red cells in the blood, but these rapidly disappear.

**Differential diagnosis.**—Care must be taken to differentiate between true icterus neonatorum (physiological jaundice) which appears on the 2nd to 4th day and is not very intense, and icterus gravis neonatorum (erythroblastosis foetalis) appearing at birth or within a few hours of birth with intense jaundice. (See p 68) Congenital obliteration of the bile ducts (see p 122) causes a jaundice which appears between the first and third weeks. Umbilical infection and syphilitic cirrhosis are rare causes of jaundice in the new born.

#### ENLARGEMENT OF THE BREASTS (MASTITIS NEONATORUM)

Enlargement of the breasts is not uncommon in both male and female infants. A secretion, popularly known as "witch's milk" may be

expressed. This condition is probably caused by some of the lactation stimulating hormone, believed to come from the pituitary gland, getting into the infant's circulation from the mother.

**Treatment.**—The breasts should be left strictly alone, and not fomented or pulled upon as is so often mistakenly done.

#### VAGINAL BLEEDING

Vaginal bleeding is probably also caused by a hormone which, being secreted by the mother, has been absorbed by the infant. This stimulates the uterus causing a transient menstruation. The condition is relatively rare, and no treatment is necessary.

#### CONGENITAL MALFORMATIONS

The incidence of congenital abnormalities among the general population is not accurately known, but careful post mortem examination has shown these abnormalities in about 15 per cent. of the infants dying at the Hospital for Sick Children, Great Ormond Street, under the age of one year.<sup>1</sup> Of those dying under the age of 4 weeks (neo natal cases) about one death in three is due to some congenital malformation.

In some cases there is malformation of a vital organ, such as the heart, kidney or bowel which is incompatible with life. In other cases the abnormality, although gross, may not involve such important organs, the child's health being unimpaired.

In a series of neo natal deaths numbering 138 studied by Spence and Miller<sup>2</sup> there were 12 deaths from congenital malformation and in 272 deaths during the first year of life 23 were due to congenital malformations. The following causes were found—

Spina bifida	Hydrocephalus	monsters etc.	11
Alimentary malformations			7
Atresia of oesophagus	2		
Pyloric stenosis	2		
Intestinal obstruction	2		
Imperforate anus	1		
Congenital heart disease			5
Mongolism			4
Mental defect			1
Osteogenesis imperfecta			1
			<hr/> 29 <hr/>

**Ætiology and pathology.**—Still<sup>3</sup> pointed out that congenital malformations are much more frequent in first born than in subsequent children. Denis Browne<sup>4</sup> accumulated a great deal of evidence to show that certain deformities are due to normal or increased intra uterine pressure exerted on a child who is in an abnormal position. He suggests that talipes equino varus, malposition of the toes, postural torticollis, displaced ears, congenital dislocation of the hip, congenital fracture of various bones, pressure scars, and dimples over pressure points, are all caused by infra-

<sup>1</sup> Donald Paterson, "Neo-natal and Post-natal Mortality," *National Health* No. 167, Aug. 1933, xvi, 36.

<sup>2</sup> J. C. Spence and F. J. W. Miller, "Report of an Investigation into the Causes of Infantile Mortality in Newcastle-upon-Tyne during the year 1939."

<sup>3</sup> *Brit. Lancet*, Oct. 15, 1932, ii, 795.

<sup>4</sup> Denis Browne, "Congenital Deformities of Mechanical Origin," *Proc. Roy. Soc. Med., Section for Study of Diseases of Children*, May 22, 1936, xlix, 1409.

uterine pressure Heredity as a cause of congenital malformations should not be forgotten There is much evidence in favour of this hypothesis Recently, Murphy<sup>1</sup> has produced figures to show that in families already possessing a malformed child the birth of a subsequent malformed child occurs with a frequency about twenty four times that of the general population Murphy concludes that gross human congenital malformations arise solely from influences which affect the germ cells before fertilization

**Variations of abnormality.**—Among the more common congenital malformations are the various types of hernia hare lip and cleft palate, club foot, congenital dislocation of the hip *nævi* congenital heart disease, congenital pyloric stenosis and congenital obliteration of the bile-ducts

**Treatment.**—In some cases, such as hare lip and cleft palate, and congenital pyloric stenosis, the treatment is surgical in others, such as heart disease and brain defect, nothing can be done in others again such as congenital laryngeal stridor, the abnormality may right it elf as the child grows older, although no specific treatment has been applied

As the majority of these malformations are described in detail under the various systems affected, it will be sufficient to give the following classifications —

- (a) *Respiratory* —Congenital laryngeal stridor (p 126) atelectasis of the lung (p 126), cystic lung (p 126)
- (b) *Cardiovascular* —Congenital morbus cordis (p 169)
- (c) *Alimentary* —Hare lip and cleft palate, oesophageal stenosis (p 90) congenital pyloric stenosis (p 90), intestinal atresia (p 89) imperforate anus (p 89), obliteration of the bile ducts (p 122)
- (d) *Nervous* —Cerebral agenesis (p 224), spina bifida (p 250)
- (e) *Skeletal and muscular* —Dislocation of the hip (p 279), Klippel Feil syndrome (webbed neck) (p 278), supernumerary digits

## BIRTH INJURIES

### INJURIES OF EXTERNAL PARTS

1 **Caput succedaneum.**—This is an oedematous swelling which forms normally in the soft tissues over the presenting part of a child's head, and it is of no clinical significance Occasionally, it hides a cephalhæmatoma It disappears without treatment in the course of a week or two

2 **Cephalhæmatoma** —This is an extravasation of blood beneath the periosteum, limited to the area bounded by the neighbouring sutures, and is due to pressure on the skull and scalp at birth Asphyxia, congestion and hypoprothrombinæmia aid its formation

**Clinical picture**—As a rule, the hæmatoma appears over the area presenting itself first during birth, occasionally, it appears over that portion pressed on by forceps during delivery It is chiefly of interest because of its tendency to persist in some cases for as much as two or three months It is most common in the occipital region where, as it is gradually absorbed, a soft mass may be felt, the sides of which are hard and irregular and

resemble a depressed fracture of the skull in the centre. Actually the sharp hard edge is caused by the elevated periosteum.

**Treatment**—A cephalæmatoma is best left untouched, a firm pad being applied in the early stages and later no dressing is needed. Aspiration involves the risk of infection and is best avoided. No permanent damage results and no further treatment is required.

**3 Hæmatoma of the sternomastoid**—This hæmatoma is probably due to the rupture of some of the muscle-fibres and vessels during birth, the sternomastoid being slightly over stretched.

**Clinical picture**—A few days after birth a lump may be felt at about the middle third of the sternomastoid or it may be noted that the child tends to keep the head tilted to one side as if a torticollis were present, attention being drawn in this way to the extravasation of blood into the substance of the sternomastoid.

**Prognosis and treatment**—Complete recovery occurs as a rule but



Fig. 4.—Baby 7 weeks old with depressed fracture of the skull at earwards successfully elevated.

occasionally slight torticollis may remain. The child should be placed to lie on the affected side to produce counter stretching. No further treatment is necessary.

#### INJURIES OF BONES

**Depressed fractures of the skull bones**—These are by far the most common of the fractures of the new born. They are actually very infrequent as the skull bones are so soft and pliable that they overlap one another and mould themselves to the various presentations. Occasionally however a shallow or deep fracture occurs after the application of forceps. The shallow ones require no treatment but the deep depressed fractures require elevation as otherwise permanent cerebral damage with after effects, tends to result. The operation is simple (Fig 4).

**Fractures of the long bones**—A fracture of the femur, humerus or clavicle may occur as a result of an unusual position of the infant or a situation requiring undue force for delivery. As a rule, healing takes place readily, and there is little or no resultant deformity.

#### DAMAGE TO THE PERIPHERAL NERVES

**Erb's paralysis.**—This is due to an injury of the brachial plexus, and is the most common peripheral nerve palsy. It is caused by stretching



Fig. 5—Erb's paralysis in an infant showing the hand held in the waiter's tip position.  
(By courtesy of Dr. Hector Cameron)

or tearing the brachial plexus during the process of birth. Acute flexion of the head puts an undue strain on the brachial plexus, just as does pulling on the shoulders with the head firmly fixed.

The clinical picture is that of flaccid paralysis, the arm hanging freely by the side, fully pronated, and the hand in the position of a waiter's hand bent to receive a surreptitious tip.

**Treatment**—If the injury be slight, and merely due to over stretching

of the nerves and possibly some hæmorrhage into the nerve sheaths, recovery takes place spontaneously in the course of a few weeks. Where, however, the injury is extensive, and the plexus has been torn across, an open operation is necessary in about 20 per cent of all cases. Meanwhile, every case should be put up in the position of rest. The arm is abducted at a right angle from the trunk, and the forearm fully supinated, the elbow flexed, and the wrist dorsally flexed. Fairbank's splint is widely used (Fig 6).

**Prognosis**—All the mild cases and some severe cases clear up completely.

**Other nerve paralyses.**—Occasionally the facial nerve is nipped by the forceps blade. This recovers without further treatment. Among rare paralyses is Klumpke's paralysis, where the eighth cervical and first dorsal roots are injured, and there is weakness of the flexors and small muscles of the hand, with no power to grip. There may be tearing of the lumbar or sacral plexus, with paralysis of the legs, or occasionally tearing or hæmorrhage into the spinal cord itself. In all these the principles of treatment are a period of complete rest, with the parts or limbs splinted in the most restful position and, if return of sensation and movement is not shown in a few weeks, open surgical operation where possible. Electrical stimulation and massage are useful adjuncts.



Fig 6.—The Fairbank splint for Erb's paralysis

#### INTRACRANIAL INJURY AND HÆMORRHAGE<sup>1</sup>

Spence<sup>2</sup> calculates that about one infant in twelve shows some cerebral hæmorrhage or contusion and that 20 per cent of the neo-natal mortality is due to this cause.

Besides intracranial hæmorrhage, less serious complications at birth, such as cerebral congestion, œdema, and simple cerebral contusion are found.

The various types of intracranial bleeding classified by Cruickshank<sup>3</sup> are as follows—

- 1 Meningeal capillary oozing. This is very common and seldom sufficient to be of any moment.
- 2 Traumatic gross intradural hæmorrhage, caused by lesions of the veins and sinuses, often associated with tears of the dural septa.

<sup>1</sup> W. S. Craig "Intracranial Hæmorrhage in the New born," *Arch. Dis. Child.*, 1924, xii, 82.

<sup>2</sup> J. C. Spence, *Neo-natal Diseases, Diseases of Infancy and Childhood*, Parsons & Darling (Oxford Med. Pub.), 1925 Vol. 1 p. 103.

<sup>3</sup> "Causes of Neonatal Death," *Medical Research Council Reports*, No. 145, 1920.



- 3 Bleeding from the choroid plexus into the ventricular system due either to trauma or to asphyxial congestion and capillary oozing
- 4 Bleeding associated with disease of the mother or of the infant. This occurs as a result of the haemorrhagic diathesis hypoprothrombinæmia or infection in the infant or of toxæmia in the mother the bleeding being usually intraventricular

Other factors influencing cerebral hæmorrhage are —

- (a) The type of presentation e.g. breech or occipito posterior presentations are more dangerous
- (b) Prematurity is often an important factor. Undoubtedly the premature infants show a high incidence of cerebral hæmorrhage and an increased tendency to bleed
- (c) Asphyxia. Where the cord is round the infant's neck and there has been partial asphyxia the tendency to ooze and bleed is much accentuated

**Signs and symptoms** — If the bleeding has been very slight or into one of the portions of the brain such as the frontal or occipital lobes where immediate signs are not obvious there may be no symptom. If however one of the great sinuses is torn across there may be a gush of blood with consequent shock, white asphyxia and death almost at once. There are several cardinal symptoms however which indicate cerebral hæmorrhage. These are —

- 1 An inability to suck well
- 2 A poor ineffective high worried cry. This is sometimes described as sounding like an animal in pain
- 3 Ineffectual swallowing
- 4 A tendency to twitch or convulse. This may be postponed as late as the third or fourth day
- 5 A tendency to lie with the eyes wide open and staring. This is quite unusual in a new born baby for any length of time. Injured infants may not close the eyes at all. The fontanelle tends to bulge suggesting an increased intracranial pressure. (For further details, see p 224)

**Prognosis** — In Fleming's 33 survivors, 5 showed symptoms of mental or physical defects. In the author's experience in those surviving after convulsions and definite symptoms of intracranial hæmorrhage at least 50 per cent later show signs of cerebral damage.

**Treatment** — The late treatment of cerebral hæmorrhage that is when the child is several months or years old is dealt with on p 226. For vitamin K treatment see p 68.

The problem in the first few days of life is largely a nursing one.

**Feeding** — As a rule, these infants are unable to suck or swallow. They therefore tend to starve. Sometimes breast milk can be expressed and given with a teaspoon or pipette but in severe cases it may be necessary to feed with an oropharyngeal tube.

**Blue attacks** — Where the colour is poor, and definite cyanotic turns occur, nasal oxygen, or better still an oxygen tent, should be used.

**Convulsions.**—If slight, one-half to one grain of chloral, given before each feed, will control the twitching or spasms. If, however, the convulsions are severe, half a grain of chloral each hour should be given until the child is sufficiently drowsy and relaxed. Amytal (Eli Lilly), one quarter of a grain, may be given from one to three times in the day, starting with the smallest dose.

Moncrieff<sup>1</sup> advocates ten times normal saline rectally, in an effort to reduce cerebral oedema which may accompany the bleeding. Two ounces of saline solution are run into the rectum slowly. This is best made up by adding one teaspoonful of salt to 2 oz. of water. The buttocks are strapped closely together to ensure that the saline is retained.

### ASPHYXIA NEONATORUM

After the birth of the infant, when respiration is established, the lungs expand slowly. Usually the anterior portions expand first and it may be two or three days before both lungs are completely inflated. It has been found that as the  $\text{CO}_2$  content of the blood rises, the respiratory centre is stimulated.

**Ætiology.**—Any cause which prevents the respiratory centre of the medulla from performing its function will produce asphyxia neonatorum. For instance, a severe cerebral hæmorrhage with increased intracranial pressure, would do this. On the other hand the infant may be premature, or have some gross respiratory or cerebral malformation. The trachea or bronchi may be blocked with mucus or blood, or the nose may be congenitally blocked or filled with debris.

Some drugs particularly morphia, when given to the mother, tend to depress the respiratory centre in the infant. The cord may be twisted or tied off, or a sub-placental hæmorrhage occur, producing intra uterine asphyxia. When these latter causes are operating the foetal heart is noticed to be slowing, and will finally cease, before birth.

**Asphyxia livida.**—In such infants there is marked cyanosis, and respiration may only commence after some time. As a rule, however, with adequate treatment, the outlook is good.

**Asphyxia pallida.**—In this type there is a condition of shock, the whole child is relaxed and pale, and the heart is beating feebly. As a rule, in such infants there is gross intracranial hæmorrhage, and the outlook is correspondingly bad.

**Treatment.**—In asphyxia livida respiration should not be stimulated by violent methods. The physician should make certain that the air passages are thoroughly cleared with a suitable rubber catheter or other suction apparatus. If necessary, the catheter must be passed down the trachea. Spanking or slapping the infant should be avoided.  $\text{CO}_2$  should be administered with a mask or funnel, or nasal catheter, and at the same time gentle pressure and relaxation of the thorax will draw the gas into the respiratory tract, thus further raising the  $\text{CO}_2$  content of the blood stream. Drawing the tongue in and out with a pair of forceps while administering the  $\text{CO}_2$  is also of value. If the infant responds slowly, a

<sup>1</sup> A. Moncrieff, "Hypertonic Rectal Saline for Intracranial Injury in the New born," *Brit. Med. Jour.*, June 16, 1934, 1, 1068.

warm mustard bath should be given. With a little patience the respiratory rhythm will be established.

Monieries<sup>1</sup> finds that, apart from those whose posterior nares are congenitally closed, some infants have dried secretions in the nostrils, and do not seem to have the knack of breathing through the mouth. Unless their nostrils are cleared out with a fine catheter they tend to be cyanosed and even to die. A drop of liquid paraffin down each nostril will keep the passages clear.

The seriously shocked infants with asphyxia pallida should be placed at once in a mustard bath and stimulants such as coramine or camphor should be given (see p. 397).

The treatment suggested for asphyxia livida should also be attempted. *Drugs*<sup>2</sup>—In 1937 a series of cases of asphyxia neonatorum was described by R. A. Wilson, at the Royal Society of Medicine. His technique was first to clear the respiratory passages of the infant, then  $\frac{1}{8}$  grain of alpha lobeline was injected into the fetal cord about 8 or 9 inches from the body of the infant, the cord being clamped off distal to the injection. The cord was then "milked" gently towards the infant and in about 15 seconds there was a sudden spasm of the respiratory muscles followed by regular respiration. The amount of lobeline given can be regulated by the amount of blood milked down the cord. Once respiration is established no further milking need be done.

The author has no great experience of this method but apparently it should be used with caution. When properly administered however it is most efficient and highly recommended.

## INFECTIONS IN THE NEW BORN

The infant inherits or acquires from its mother a temporary passive immunity to various infections particularly measles and diphtheria. It does not seem, however, to acquire immunity to streptococcal or other pyogenic infections. It does not appear to be able to localize its infections as can older children and adults by stopping the infection at the appropriate gland which drains the infected area. Septicæmia is therefore extremely common, and probably often overlooked.

**Modes of infection**—1 Infection may pass to the infant in the uterus, through the placenta. Examples would be syphilis, mumps, and typhoid fever.

2 During birth the infant may become infected from the liquor or the maternal passages. This is seen in *ophthalmia neonatorum*.

3 The commonest time of infection is after birth. The umbilicus acts as a frequent portal of entry. The infant's skin however, is very apt to become infected but any part of the body such as the lungs, kidneys and alimentary tract may be affected. Many cases of infection by the *hemolytic staphylococcus* from a nurse have been recorded. In turn, the infant is said to infect the mother's nipple, producing a breast abscess. *Pemphigus neonatorum* may result from such an infection.<sup>3</sup>

<sup>1</sup> A. Monieries, "Nasal Obstruction in the New-born," *Brit. Med. Jour.*, June 27 1936, 1 1295.

<sup>2</sup> R. A. Wilson, "Treatment of Asphyxia Neonatorum with Alpha lobeline," *Proc. Royal Soc. Med.* April 16, 1937, xxxi 1461.

<sup>3</sup> D. D. Elliott, E. H. Elliott and F. Holland, *Lancet* 1941 1 267.

**Sepsis neonatorum**—Shortly after birth an infant may show signs of ill health. The site of the infection may be obvious as for instance when the skin is infected and lullæ result. On the other hand, commonly nothing is seen externally and the explanation may only be found *post mortem*.

**Umbilical infection.**—Probably the umbilicus is the original source of infection in a large number of cases. The cord does not dry up and drop off or separate as it should. Thick creamy pus may exude from it, particularly when the abdomen is stroked from the liver toward the navel. In such cases the round ligament with the obliterated umbilical vessels will be found heavily infected and the source of this pus. More often than not however nothing is seen on examination beyond a slight moisture of the navel and it must be assumed that the infant has a septicæmia.

**Clinical picture**—In the new born infant an infection in any part of the body tends to produce a symptomatic diarrhoea. The infant tends to become dehydrated and in a small proportion of cases jaundiced from an infected liver. The skin may show widespread lesions and the condition known as exfoliative dermatitis is seen. Pneumonia may occur and peritonitis, empyema and meningitis are all found. An examination of the urine may show some albumin or pyelitis may be present. The temperature is not of diagnostic value as it may be normal high and swinging, or intermittent and its height is not a gauge of the severity of the condition.

**Treatment**—Careful and expert nursing is of the greatest importance. Where possible breast milk should be given. The sulphonamide preparations should be exhibited in suitable cases (see pp 888-895). An intravenous saline and glucose drip is necessary where dehydration is marked and feeding by a stomach tube may be necessary.

**Erysipelas**—This condition occurs in new born infants (see p 297). Sulphonamide treatment has greatly lowered the mortality.

**Diphtheria.**—This is rare in infants before the age of one year probably because they have inherited immunity from the mother. See p 819.

**Scarlet fever, Measles and Rubella** are all extremely rare before the age of 6 months to 1 year (pp 807, 811-810).

**Whooping-cough and Chicken-pox**—Occasionally these occur in the neo-natal period (pp 815-819).

**Thrush**—The seriousness of this infection has undoubtedly escaped attention. Ebbs' recent paper on oesophagitis due to it is important. (For details see p 96.)

**Neo-natal pyelitis**—This condition is very much commoner than is at present realized and males are infected almost as often as females at this age. The symptoms are usually a failure to thrive and restlessness with diarrhoea. A careful examination will show infected urine. The temperature is high and continuous. For treatment see p 202.

#### TETANUS NEONATORUM

(TRISMUS NEONATORUM OR LOCKJAW)

**Ætiology and pathology**—This disease is much less common now than formerly. It was at one time ascribed only to infection on of the umbilicus by the tetanus bacillus the toxins acting at a distance on the brain and spinal cord. Inoculation of scrapings

from the umbilical cord into guinea pigs has produced the disease. Some authors hold that the same clinical picture can be produced by other organisms such as the streptococcus producing a meningitis from which the patient may recover.<sup>1</sup>

**Clinical picture.**—On the third to fifth day the child shows a tendency to sudden spasm. The hands and jaws are clenched and any attempt to force the jaws apart results in a sudden spasm of the whole infant. The nourishment of such an infant is very difficult. Spasms interfere with respiration, and death, if it occurs, is due to general exhaustion.

**Prognosis and treatment.**—The majority of cases are fatal. Occasionally, however, if they are treated early and given antitetanic serum, both subcutaneously and intrathecally, recovery takes place. A relatively large dose of serum is required (see p. 235).

### OPHTHALMIA NEONATORUM

**Ætiology.**—The commonest cause of ophthalmia is the gonococcus, the disease being contracted as the child passes through the infected maternal passages. Occasionally other organisms, such as the pneumococcus streptococcus or *B. coli* are the cause.

**Clinical picture.**—On the second or third day the eyes become red, swollen and begin discharging. A short acute course is run and in three or four weeks the infection has settled, leaving a greater or lesser amount of damage behind depending on the treatment. Ulceration of the cornea is the commonest complication with perforation and then destruction.

All ophthalmic discharges during the first 21 days of an infant's life are notifiable and 70 per cent. of such discharges have been found to be due to the gonococcus.

**Treatment.**—In the Metropolitan area there is a special hospital for ophthalmia neonatorum—St. Margaret's, Kentish Town, where the mothers of the patients are admitted and breast feeding is continued. The routine treatment is irrigation of the eyes with a solution of eusol (1 in 7) at intervals of one, two or three hours according to the extent of the discharge. After each irrigation one drop of a solution of acridavine in castor oil (1 in 1,600) is instilled. Since the introduction of sulphonamide and M & B 693, the treatment has improved greatly. One-quarter to one-third of a tablet each four hours day and night, for three days or more is recommended.

**Prophylaxis.**—A weak silver nitrate solution (say, 1 per cent.) or a 5 per cent. solution of argyrol, instilled into the eyes at birth should act as an efficient prophylactic.

**Prognosis.**—If taken early and properly treated the outlook is excellent (see pp. 383, 388).

### PEMPHIGUS NEONATORUM<sup>2</sup>

#### (BULLOUS IMPETIGO)

**Ætiology and pathology.**—Staphylococcal infections of the skin occur most frequently in early infancy, and may often be contracted from the infected finger of the maternity nurse or a cracked nipple in the mother. The infection spreads rapidly over the infant's body, forming large bullæ containing thin pus (Fig. 7). Delay in treating these may prove fatal.

Epidemics occur in institutions and may often be traced to one common source. The organism is usually the hemolytic *Staphylococcus pyogenes aureus* (see pp. 296, 385).

**Treatment.**—At the first signs of infection the infant should be removed from contact with other infants and the source of the infection sought. Blebs of any size should be clipped, allowing the pus to drain, and the whole carefully powdered and kept as dry as possible. If the infection is extensive it will be found best to place the child in a warm bath of some weak antiseptic, such as Condy's fluid, several times daily drying and powdering afterwards. A mixture of zinc oxide and starch powder equal parts, will be found most useful. Boracic powder should be avoided as being hygroscopic and tending to keep the child moist. Care should be taken to continue nourishing the infant as well as possible. Strapping with zinc oxide elastoplast is most beneficial. The mortality in extensive cases remains very high. One of the sulphonamide preparations, such as M & B 693 or sulphathiazole (M & B 760) may be

<sup>1</sup> H. C. Cameron, *Ory's Hospital Gazette* Dec. 20, 1922, xxxvi, 43.

<sup>2</sup> *Lancet*, Feb. 15, 1937, i, 897.

given—one quarter to one third of a tablet each four hours, day and night, for three or four days. Recent experience has shown that sulphamethazine is the best of the sulpha preparations for the purpose.



FIG. 7. Case of pemphigus neonatorum.

## BLOOD ABNORMALITIES

The various blood diseases are discussed in Chapter V, p 173. Physiological icterus neonatorum has been described on p 57. Among the rarer conditions are those described below.

### HEMORRHAGIC DISEASE OF THE NEW BORN (HYPOPROTHROMBINÆMIA)

**Incidence.**—This disease occurs in 0.5 to 1 per cent of all infants. It appears to be due to two factors, an absence of vitamin K, and trauma sufficient to interfere with the integrity of the vascular walls. All normal infants show a lowering of their prothrombin index between the first and seventh day. The indices of some infants fall to 20 per cent of the adult level and bleeding occurs from any trauma. A clotting time of 8 to 16 minutes or longer is most significant.

**Sites of hæmorrhage.**—The commonest site is from the gastro-intestinal tract, giving rise to so called melæna neonatorum. There may, however, be vomiting of blood. Other sites are intra-cranial after asphyxia or trauma, and bleeding into the suprarenal. When there is melæna neonatorum an apparently healthy infant becomes flaccid on the second or third day or later and passes large tarry stools filled with blood clots. Collapse and symptoms of hæmorrhage are present. *Post mortem*, nothing is found beyond petechial hæmorrhages into the gastro-intestinal wall. An infant may suck blood from the mother's cracked nipple, and both vomit and pass blood in the stool. This possibility should be excluded before a diagnosis of melæna neonatorum is made.

**Treatment.**—A blood transfusion should be given when possible, otherwise, 10 to 20 c.c. of adult whole blood should be given sub-cutaneously and at the same time 2 to 5 mgm. of vitamin K, 2-methyl-1,4-naphthoquinone-hapilon (Glaxo) or Synkavit (Roche Products). This is best given subcutaneously, and can be repeated daily for a few days by mouth.

**Prophylaxis.**—10 mgm. of Vitamin K given to a woman at the commencement of labour appears completely to protect her infant from hypoprothrombinæmia.<sup>1</sup>

### HEMOLYTIC DISEASE OF THE NEW BORN (ERYTHROBLASTOSIS; ICTERUS GRAVIS NEONATORUM; HYDROPS FETALIS)

This is a form of hæmolytic anæmia affecting new born infants. The majority show well marked jaundice at or shortly after birth (icterus gravis). A very few are

<sup>1</sup> E. G. Poncher. *The Role of Vitamin K in Hemorrhage in the New Born Period. Advances in Pediatrics* Vol. 1. Helmhann 1942.

A. I. S. Macfarlane, L. McCallum and W. T. Haultain. *The Effect of Intra-partum and Neo-natal administration of vitamin K analogues on the newborn. Brit Med Jour* 1940 1: 893.

oedematous and are usually still born (congenital hydrops foetalis) and a few show anaemia, without jaundice or oedema

**Ætiology and pathology**—Landsteiner and Wiener<sup>1</sup> demonstrated in 1941 that the red cells usually contain an agglutinin which they named Rh because a similar agglutinin is present in the red cells of Rhesus monkeys. They showed that this agglutinin is distributed amongst different persons irrespective of their A/B/O group and that amongst American whites only about 15 per cent of persons lacked agglutinogens, i.e. were Rh negative. Wiener and Peters<sup>2</sup> showed that this 15 per cent (Rh negative) whose erythrocytes lack the Rh agglutinin, may develop specific anti Rh agglutinins if they are transfused with blood containing the Rh agglutinin (Rh positive). Levine, Katzin and Burnham<sup>3</sup> (1941) advanced the iso immunisation theory—a man whose blood contains the Rh factor mates with a woman whose blood does not contain the Rh factor, i.e., who is Rh negative. If her foetus is Rh positive she may produce anti Rh agglutinins as a result of immunisation with the foetal blood. The antibodies pass through the placenta and in suitable concentration cause hæmoly sis of the foetal red cell. The consequent anaemia calls forth a great effort of response from all erythropoietic tissues (Gimson, 1943<sup>4</sup>).

The blood shows a varying degree of anaemia and the hæmoglobin may be as low as 20 or 30 per cent, with a corresponding reduction in the red cells. A differential count shows a very high proportion of nucleated and other immature red cells. The liver and spleen are enlarged and *post mortem* all extra medullary, as well as medullary tissues show extreme hæmopoietic activity. The Van den Bergh shows a strong indirect reaction suggesting that the jaundice is hæmolytic in origin but the reaction may be bi phasic and show a prompt direct reaction due probably to the plugging of the bile canaliculi by the products of hæmolysis.

The urine contains blood pigments, and the faeces contain bile

**Clinical picture**—Often at birth, or within the first 48 hours an infant develops severe jaundice. Only 3 out of Gimson's 19 cases were first children the remainder were second, third or fourth in the family, with a family history of miscarriages still births, premature and previously jaundiced infants. Occasionally the first child had been quite normal. Anaemia rapidly develops, along with the jaundice and death takes place in a high proportion of untreated cases. An examination of the blood shows the picture described above. The infant sucks and feeds badly fails to thrive and rapidly deteriorates.

**Treatment and prognosis**—The infant should be given as soon as possible a blood transfusion with Rhesus negative blood free of agglutinins. The formula for calculating the amount of blood required as recommended by Gimson is

$$\frac{\% \text{ rise of Hb required}}{100} \times \text{blood volume (i.e., 40 c.c. per lb. body weight)}$$

e.g. In an 8 lb. baby, who has a Hb. of 30%

$$\frac{70}{100} \text{ (i.e. rising from 30\% to 100\%)} \times (8 \times 40 \text{ c.c.}) = 224 \text{ c.c.}$$

Therefore, amount of blood required for transfusion 224 c.c.

The transfusions are given intravenously, by drip, into the internal saphenous or a cubital fossa vein, and a constant rate of 15 to 20 c.c. per hour should be maintained. Further blood counts should be done, and a second transfusion given if necessary.

Patients who are normal mentally and show no signs of cerebral damage at the time of the transfusion appear to make a complete recovery. In Gimson's series of 19 cases 16 appeared normal, two were spastic mental defectives, and one showed kernicterus.

It is necessary to keep (and renew frequently) a store of Rh negative blood (group O) in all children's hospitals and maternity hospitals.

<sup>1</sup> K. Landsteiner and A. S. Wiener *Jour. Exp. Med.* 1941, lxxiv, 309.

<sup>2</sup> A. S. Wiener and H. R. Peters, *Ann. Intern. Med.* 1940, xlii, 2306.

<sup>3</sup> J. Levine, J. M. Katzin and J. L. Burnham, *Jour. Amer. Med. Assoc.*, 1941, cxvii, 843.

<sup>4</sup> J. Gimson, "Hæmolytic Disease of the New Born," *Brit. Med. Jour.*, Sept. 4th 1943, ii, 791.

P. L. Mollison, *Lancet*, Roy. Soc. Med. N.S. 1943, xxxvi, 441.

## CHAPTER VI

### DISEASES OF NUTRITION

#### VITAMINS

The student may well consult the work of Hopkins (1912)<sup>1</sup> and Mellanby (1921)<sup>2</sup> in England and of McCollum (1927)<sup>3</sup> and Hess (1930)<sup>4</sup> in the United States for description of the early work on vitamins. He will find excellent summaries in the book by L. J. Harris<sup>5</sup> and in the Medical Research Council's Survey.<sup>6</sup>

Vitamins may be defined as substances which are essential for normal health. Deprivation results in complete cessation of growth. As a result of deprivation diseases develop which are termed deficiency diseases. This theory was first put forward by Funk in 1912 and has since been proved to be correct.

Vitamin A (or fat-soluble A) appears to be necessary for normal growth and has a specific action on epithelial tissue: the normal coverings of the body and the mucous membrane lining the important cavities and ducts of the glands. It is a yellow pigment known as carotene which when absorbed produces vitamin A in the liver. Carotene is found in carrots, green vegetables, milk fat, fish liver oil such as cod, halibut or tuna, and in butter and eggs, liver and kidney. Heat does not destroy it. It is absent from vegetable oil. Vitamin A is stored in the liver.

One pint of dried milk is said to contain 1 000 I.U. The exact number of units necessary for health is not definitely known (Blackay). Half a teaspoonful of cod liver oil contains as a rule 1 000 units. Infants probably require 2 000 I.U. and older children from 3 000 to 5 000 I.U. daily.

Since Vitamin A is necessary for the formation of visual purple, found in the rods and cones of the retina, its absence produces night blindness. This vitamin is also necessary for the maintenance of columnar epithelium and in its absence the epithelium undergoes metaplasia and keratinisation. This produces xerosis of the conjunctiva (xerophthalmia) and ulceration of the cornea (keratomalacia). The epithelium of the respiratory system, renal system, to a lesser extent the alimentary tract, and also the follicles of the skin are affected. The skin over the outside of the arms, particularly the upper part, may become dry and rough and is often called "toad skin".

Carotene may be present in the diet but not absorbed, e.g. when an excess of mineral oil (liquid paraffin) is given by mouth, as carotene is soluble in liquid paraffin. In a condition such as coeliac disease, where there is poor fat absorption, carotene may be lost in the faeces, along with the remainder of the intestinal fat.

<sup>1</sup> F. G. Hopkins, *Journ. of Physiol.* 1912, xlv, 4-5.

<sup>2</sup> E. Mellanby 1921 Medical Research Council's Special Report series, No. 61, London.

<sup>3</sup> E. V. McCollum and W. S. Steenbock, 1929, *Newer Knowledge of Nutrition*, 3rd ed., New York.

<sup>4</sup> A. P. Hess, *Rickets* 1930, London.

<sup>5</sup> L. J. Harris, "Vitamins in Theory and Practice", Cambridge Univ. Press, 1935.

<sup>6</sup> Medical Research Council, "Vitamins—a Survey of present Knowledge", H.M. Stationery Office, 1937.



TABLE XII  
REQUIREMENTS OF VITAMINS (*Med Clin N. Amer.*, 1943, xxvii, 280)

Age	A			B		C		D		E		K	
	I U.	Mg	I U.	Mg	I U.	Mg.	I U.	Mg of D*	I U.	I U.	Mg	I U.	Mg
Adult ..	5,000		600	1.8	1,500	75	300(?)—600(?)*	0.0075—0.015	No international unit	No international unit	Unknown	No international unit	0.001(?) for new-born
Under 1 year ..	1,500		125	0.4	600	30	100—800	0.01—0.02					
1-3 years ..	2,000		200	0.6	700	35	400—800	0.01—0.02					
1-6 years ..	2,500		265	0.8	1,000	50	3,500	0.0075					
7-9 ..	3,500		373	1.0	1,200	60	weekly	weekly					
10-12 ..	4,500		400	1.2	1,500	75	300(?)—	0.0075—					
Girls 13-15 years ..	5,000	Unknown	466	1.4	1,600	80	400(?)*						
Boys 10-20 ..	5,000		400	1.2	1,400	80							
Boys 13-15 years ..	5,000		532	1.6	1,800	90							
Boys 10-20 ..	5,000		606	2.0	2,000	100							

\* It is not certain whether adolescents and adults need an extraneous supply of vitamin D. Enough may be made under the skin by the action of light.

*Requirements and Body Weight*—Vitamin A Requirements directly proportional to body weight and not age. Vitamin B<sub>1</sub>. Requirements depend on caloric intake and not body weight. Vitamin C Requirements depend upon degree of body metabolism rather than on actual body weight, e.g., a small person suffering from pyrexia will need more than a larger person who is asexual. Vitamin D. No apparent relation between body weight and requirements.

Vitamin A has been described as the 'anti-infective' vitamin but it is not. Undoubtedly if the mucous membranes are not intact, through deficiency of this vitamin they allow infections to enter, but there is no anti-bacterial action.

Xerophthalmia appears first as dry, rough, yellowish white patches on the bulbar conjunctiva (Bitot's spots) these are small and triangular and usually situated at first on the nasal side. It is not practicable to test very small children for night blindness and there is no way of confirming the diagnosis scientifically.

*Treatment*—Night blindness shows rapid improvement under treatment. Halibut liver oil 10 drops three times daily contains many thousand units of vitamin A and this should be considered a curative dose. Spence (1931) found in his 17 cases that 10 to 15 c.c. of cod liver oil daily rapidly produced a cure. If the vitamin is not being absorbed it can be given subcutaneously. Helen Mackay's series of infants suggested that there was sufficient vitamin in the ordinary infant's diet. The tendency at Welfare Clinics to give synthetic vitamin D without adding A should be deprecated. Natural sources such as cod liver or halibut oil are on the whole safer.

**Vitamin B Complex** is made up of various chemical substances, the absence of which produces a number of clinical pictures some of the better known of which are beriberi, pellagra and cheilosis. The foods which contain a rich supply of this vitamin are wheat germ, yeast, extract, eggs, meat (particularly liver) and leafy vegetables also, to a lesser extent, fruit. Nuts, bran, pulses, pork and whole meal bread also contain this complex. (For its action in coeliac disease see p. 84.)

**Vitamin B<sub>1</sub> (Aneurin or Thiamin)** is a substance the absence of which produces beriberi, a form of polyneuritis with cardiac dilatation, rapid heart's action and finally death with oedema. It is claimed that early stages of vitamin B<sub>1</sub> deficiency are indicated by lack of appetite, digestive upsets and failure to gain weight.

Chemical investigations have not shown any proof that there is a widespread shortage of this vitamin, particularly since the institution of the National Loaf. As aneurin or thiamin has been shown to be necessary for the proper metabolism of carbohydrate, a liberal supply is undoubtedly advisable. The requirement for infants appears to be from 0.25 to 0.6 mgm daily and for children from 1.0 to 2.0 mgm daily. (One mgm equals 1/1000 lb.) One pint of milk contains 120 I.U. or is sufficient for an infant's daily requirements of B<sub>1</sub> alone.

**Vitamin B<sub>2</sub> (Riboflavin or Lactoflavin)** is a substance which can be isolated from milk, eggs, liver, yeast, leafy growing vegetables, fish, roe and kidney. Children from 3 to 12 years require from 1 to 2 mgm daily. A deficiency of this vitamin causes cheilosis which is characterised by sores about the corner of the mouth and under the nose, also keratinisation of the cornea. Probably angular stomatitis (perleche) described by the French, is the same deficiency disease.

**Nicotinic acid P.P. (Pellagra Preventive)** factor is not destroyed by ordinary cooking. It is sometimes called Niacin and it may form

Dichlorophenolindophenol (British Drug Houses) may be used as a simple method for ascertaining the presence of ascorbic acid in the urine.<sup>1</sup>

**Vitamin D (or fat-soluble D)** The two most important compounds with anti rachitic properties are vitamin D<sub>2</sub> (irradiated ergosterol or cholecalciferol) found in vegetables and D<sub>3</sub> (7 dehydrocholesterol) found in animal fats. This vitamin can be formed in the fat cells of the skin by the action of the ultra violet rays of the sun and rickets will not develop unless both the anti rachitic vitamin of the diet is deficient and at the same time the ultra violet rays are absent. Vitamin D has a specific action on cartilage and its calcification and is usually found in association with fat soluble A.

**Sources**—Some of the best known sources are milk, cream, butter, egg yolk, cod liver oil, halibut liver oil and other fish oils. Vitamin D<sub>2</sub> (calciferol) the synthetic preparation is available for those who cannot tolerate fish liver oil or where large concentrated doses are necessary. (See p. 405) Government Cod Liver Oil has a minimum vitamin D potency of 200 I U per gramme i.e. 12 I U per minim.

**Requirements**—An infant premature or otherwise or a young child requires about 700 I U per day i.e. 60 minims or one teaspoonful of drichum cod liver oil which would contain 720 I U.

### DEHYDRATION FEVER (INANITION FEVER)

Any infant kept short of fluid for a few hours tends to develop a fever. For instance in the first three or four days of life, if the colostrum is scanty and the milk slow in appearing unless frequent drinks of water are given especially in summer time the child becomes progressively dehydrated and runs a temperature of from 100° to 102°. This subsides at once on the administration of sufficient fluid and can be prevented by giving drinks of boiled water from the age of 12 hours onward until a sufficient flow of milk has appeared.

Dehydration fever may be seen at all ages. Part of the fever present in acute dietetic diarrhoea is undoubtedly due to deprivation of fluids. Every infant should be offered from 4 to 6 oz. of water daily in addition to its ordinary feeds and if any considerable proportion of this is taken, the feed should be reconsidered being probably too small in bulk and calling for further dilution (see pp. 23-57).

### MARASMUS (WASTING INFANTILE ATROPHY)

**Clinical picture**—Frequently, the practitioner is asked to see an infant who is said to be wasting. The infant although several weeks old has remained at its birth weight or may have gained well at first and recently the gain has ceased. There may, however, have been an actual loss of weight. Such an infant has an old man appearance. The skin is wrinkled and the sucking pads stand out prominently on the cheeks. The thighs and buttocks show folds in the skin. The child may be bright and intelligent, but presents all the appearance of wasting or starvation. There may or may not be diarrhoea and vomiting.

<sup>1</sup> D. P. Hater. Estimation of True Ascorbic Acid in Urine. *Lancet* June 26 1943 1 80.

<sup>2</sup> J. J. Harris, Vitamin Saturation Test, *Lancet* April 24 1943 1, 815.

<sup>3</sup> Dosage of Vitamin D (Report of Sub-Committee—British Paediatric Association) *Arch. Dis. Child.* March 1943 xvi, 28.

In such cases a blood transfusion or two will be found useful and sometimes starts a gain in weight. At a stop, nothing abnormal may be found.

### RICKETS

Rickets is a deficiency disease due to a lack of the fat-soluble vitamin D.



Fig. 8 Severe rickets showing enlargement of the wrists and ankles and curving of the tibiae.  
(By courtesy of Dr. Victor Cameron)

and/or an insufficient intake of calcium and phosphorus in the diet leading to an imperfect calcification of the bones most obvious at the sites of growth.

**Age-incidence**—The most active signs of rickets appear between the ages of six months and two years but there seems no reasonable doubt that rickets is in process of development almost from birth onward. The florid manifestations appear to be held off for at least three months and to be uncommon before six months.

**Clinical picture.**—Bony manifestations of rickets.—Low pronounced rickets.—The head is large and large. The teeth are late—often none appear until after one year of age—and when they do come the enamel is poor and caries takes place early. The bones of the legs and arms bend with pressure so that the tibia and femora are bent either in or out giving rise to knock knee or bow legs. The forearms are curved because the child supports its weight on them when sitting (Fig 8). The anterior fontanelle is normally closed at about one year to 15 months in rickets it remains widely open even up to the second year.

**Deformities of the chest**—Harrison's sulcus is a groove running round the chest at the attachment of the diaphragm the pull of the diaphragm on the soft ribs having drawn them in at this point causing

the splaying out or eversion of the lower edges of the ribs. At the costochondral junction beading is found and these enlargements are as prominent on the inside of the rib as on the exterior. The back is bent either kyphosis or scoliosis being present. This is due to muscular weakness however as the curve is easily undone if the child be picked up beneath the arms or placed on its face.

As a rule during the active stage of rickets pallor and anaemia are common but there are exceptions to this. Sweating of the head is constant and may be most striking. The muscles generally are soft flabby and diminished. There is a tendency to indigestion and constipation of the stomach.



Fig 9—Severe rickets showing the typical chest of the greyhound large abdomen and square philosopher's head  
(By courtesy of Dr Hector Cameron)

and bowels. The old saying that the rickety child has the head of a philosopher, the chest of a greyhound, the legs of a grand piano and the "tummy" of a poisoned pup is extremely apt (Fig 9).

**Craniotabes**—In the young infant accompanying other manifestations of rickets there is sometimes gross thinning of the skull bones, so that on slight pressure they give way with a feeling like pressing on parchment. This is known as *craniotabes* and premature infants and the poor one of twins often suffer in this way.

**Nervous manifestations** (**LOW CALCIUM RICKETS**)—Hyper excitability of the nervous system giving rise to certain symptoms may appear in a case of acute rickets.

**Tetany (spasmophilia)**—Tetany manifests itself by—

1 *Facial irritability* or *Chvostek's sign*—When the cheek is tapped so that the facial nerve is irritated the muscles innervated by this nerve pass into spasm. The whole face gives a well marked twitch on that side.

2 *Carpopedal spasm* or *Trousseau's sign*—The hands are held in the accoucheur's position with the fingers fully extended but slightly flexed on the palm and the thumb adducted (Fig. 10). The toes assume much the same position as the hands. There seems no doubt that the child is in pain and suffers from cramps of the hands and feet. The typical position may be brought about in mild cases of tetany by gentle pressure on the forearm so that the hand becomes congested.

3 *Laryngismus stridulus* (crowing)—A spasm of the larynx occurs whenever the child is suddenly roused, frightened, crossed or annoyed. The larynx is held, the face becoming blue much as during a whoop in whooping cough, then it relaxes and a long drawn inspiratory cry is heard. This

is a dangerous condition and sometimes proves fatal.

4 *Convulsions* (see p. 274)—Children with acute rickets and tetany are very liable to convulsions which often occur so frequently that the child passes from one convulsion to another. There is no way of distinguishing these from early epileptic fits except by noting the other manifestations of active rickets.

**Ætiology**—Active rickets or *spasmophilia* is characterized by a low blood calcium which normally should be about 10 mgm per cent. In *bony rickets* the blood phosphorus is deficient being less than the normal which is 5 mgm per cent. The plasma phosphatase, which is normally 10 to 20 units up to 2 years of age, may be raised in active rickets up to 50 or 60 units or more. This is a very sensitive and accurate test for active



Fig. 10. Carpopedal spasm in tetany.

**rickets** In turn the calcification of the bones is deficient so that there is actually less calcium in the bones than in the normal child. Two of the factors bringing this about are

1 *Lack of fat soluble vitamin D* (see p. 74) —The absence of this vitamin may be due to diet deficiency or to lack of sunshine. The rays of the sun by acting on the skin have the power to produce the fat soluble vitamin D in the fat globules of the child's skin and thus to promote calcium and phosphorus deposition as efficiently as if the vitamin were given in the food. A vitamin deficient diet in the presence of sunshine might be tolerated, but the same diet in the absence of sunshine would produce gross rickets.

2 *Calcium deprivation* —Even in the presence of sufficient vitamin and sunshine rickets will result if the diet is so poor in calcium and phosphorus that too little is available for the needs of the body. Usually a pint of cows' milk daily provides sufficient of such minerals. Some of the sweetened condensed milks are so deficient in protein and calcium and contain so much carbohydrate that rickets results from their use.

There is present in wholemeal flour and oat flour a substance (phytic acid) which has the power to precipitate and combine with calcium making it unavailable for use by the body. Additional calcium has therefore been added to National Flour to prevent this. Farinaceous proprietary foods administered with too little milk tend to produce rickets because of their phytic acid content.

**Pathology** —There is a failure of lime deposition in the multiplying cartilage cells of the epiphyses and also in the flesh forming bone cells. Normally each maturing cartilage cell gives rise to another and ultimately calcification and ossification take place. In rickets this process becomes disordered and the line of calcification is replaced by much cartilaginous and vascular tissue. When healing takes place a new line of calcification in the middle of the metaphysis can be seen and ossification follows this process.

The proliferation of the cartilage can be seen clinically by an enlargement at the costo-chondral junctions and the wrists and ankles.

**Diagnosis** —The clinical diagnosis of rickets is extremely difficult in slight cases. Since the bony malformations remain long after the condition has healed, the clinician is apt to diagnose rickets in cases where it is already cured. If nervous manifestations are present in addition however, the condition is active. An examination of the blood calcium or phosphorus is difficult and expensive. There is no way so certain as by examining the epiphyses with the X rays, and a radiogram of the wrist shows clearly whether the child has rickets. (Plate 1.)

**Prognosis** —Once the bony deformities are present in marked degree they remain to a greater or lesser extent for some years. If the rickets has been allowed to continue stunting and dwarfing tend to occur. The nervous manifestations however, leave no residuum so far as is known. The prognosis for life is excellent though death occasionally results from laryngismus stridulus or from some acute infection engrafted on florid rickets. Rachitic infants withstand infection extremely badly.

**Treatment.**—1 *Dietetic*—The diet should contain a sufficient quantity of (a) animal protein, (b) animal fat, (c) calcium. To ensure this, no great excess of starchy food should be given. The recent discovery that wholemeal flour contains phytic acid<sup>1</sup>, which precipitates available calcium in a form which is unavailable for the body's use may make it necessary to add calcium to such starchy preparations. Up to 6 months of age, at least one pint of milk is necessary daily to supply protein, fat and calcium. After this age, eggs and bacon fat and later fresh meat, fish and chicken should be introduced in addition to the milk. (See pp. 43-44 for suggested diets.)

2 *Medicinal*—Whenever possible vitamins should be given in their natural form. It is best therefore in supplying vitamin D, both for the prevention and cure of rickets, to give it as cod or halibut liver oil, rather than in synthetic preparations. It is generally accepted that the maintenance dose of vitamin D is 700 I.U. but double this dose or more could safely be given as treatment. Since Government cod liver oil has a vitamin D content of 12 I.U. per minim a drachm or teaspoonful (60 minims) should prevent rickets in the infant or child if given each day. (For full table of vitamin D values see Appendix p. 105.) It is necessary to give vitamin D to both breast fed and artificially fed infants. Where cod liver oil is not tolerated one of the concentrated preparations such as halibut oil or synthetic preparations such as Calciferol, Ostein or Radiostoleum may be substituted.<sup>2</sup>

Premature infants require from one to two thousand units as a prophylactic dose daily commencing at the 10th day and continuing until they have regained normal weight. In cases of tetany up to 15 grains of hydrated calcium chloride or 20 grains of calcium gluconate should be given three times daily by the mouth together with a suitable dose of vitamin D. Both these salts can be given intravenously, in sterile solution and in much smaller quantity in an emergency.

3 *Sunshine*—The deliberate exposure of infants to sunshine is necessary in the British Isles. Since this is often not possible, because of weather conditions artificial sunlight can be given with great benefit, both as prophylaxis, and certainly as treatment, particularly in the winter months. A good working rule is to insist on 4 hours fresh air out of doors, per day for every infant.

**Late rickets (vitamin-resistant rickets).**—As a rule, infantile rickets is cured by the age of 2½ years and it is very rare indeed to see a case of active rickets after this age. Occasionally, however, an older child shows marked knock knee with obvious enlargement at the ankles and wrists, and an X-ray and blood phosphatase estimation<sup>3</sup> bear out the diagnosis. It will be found that a proportion of these cases is particularly resistant to treatment with vitamin D. It may, in fact take many months to get the rickets to heal. In the author's experience plain cod liver oil will cure such cases better than calciferol or other synthetic preparations. No reasonable explanation can be offered for this failure to heal. Vitamin D

<sup>1</sup> Review of Phytic Acid and Calcium Absorption. Editorial, *The Lancet* Feb. 150 1943 I, 211.

<sup>2</sup> *The Incidence of Rickets in Harrogate*. Report of the British Paediatric Association to the Minister of Health (*Reports on Public Health and Medical Subjects* No. 9" H.M. Stationary Office 1941).





Plate 1—Radiogram of wrist in a case of severe rickets



Plate 2—Radiogram in a case of infantile scurvy showing a subperiosteal hæmorrhage at the lower end of the femur

(By courtesy of Dr Hector Cannon)

**Pathology.**—Due to the deficiency of this vitamin C there are universal hæmorrhages into the growing ends of the bone, into the gums, kidney, co-sto-chondral junctions beneath the periosteum into the bone marrow itself and subcutaneously especially about the eyes (Fig. 11) also into the joints.

**Symptoms**—The mother notices that the infant is unhappy especially when anyone approaches it and attempts to pick it up. When left perfectly still the child seems less anxious. Movement, therefore, is painful. The limbs are kept still and the child appears pale and fails to thrive. Should treatment be withheld purpura next appears in the skin especially about the eyelids. An examination of the blood shows the platelets to be quite normal. Swellings appear over the shins, knees or thighs and X rays show these to be large subperiosteal hæmorrhages (Plate 2). Later, the X ray of the growing ends of the bones will show a typical white line. The



Fig. 11 Case of scurvy showing orbital hæmorrhages

urine is red or smoky and a microscopic examination shows this to be due to the presence of red blood corpuscles. Blood is usually present in the urine of scorbutic patients sometimes in microscopic amounts only. At this age there are usually two lower incisors and about the base of these will be seen purple hæmorrhagic areas and the gums are swollen. An examination of the ribs shows that the sternum has become depressed and dislocated at the co-sto-chondral junction—that is, passing the hand out from the sternum towards the ribs the depression is reached then the sharp tip of the rib is felt. This is quite unlike the beading in rickets which is a smooth oval beading with slight depression at either side and not only at the sternal side as in scurvy (Fig. 12.)

**Diagnosis**—Children who appear moderately well nourished and cry incessantly may be thought to be suffering from acute osteomyelitis especially if the swellings are large and accompanied by some infection

producing a fever. If the purpura is extensive the case may be thought to be one simply of purpura haemorrhagica or if one limb is specially involved scurvy may be mistaken for traumatic dislocation of the epiphysis or a fracture or sarcoma. Where the hæmorrhages are not very apparent, such diagnoses as tetanus, or earache are commonly made. The clinical picture, however, is a very definite one. hæmorrhages about the gums, tenderness of the limbs with or without swellings, blood in the urine, backward dislocation of the sternum at the costochondral junction and hæmorrhage beneath the periosteum, the hæmorrhages being confirmed by X rays.

**Prognosis.**—Properly treated all cases of scurvy should recover completely. When the case is complicated by some infection scorbutic infants resist the infection badly and the prognosis becomes less favourable.

**Treatment.**—Vitamins in the form of orange juice, grapefruit juice, or tomato juice, should be given. Quantities of the latter equivalent to from one to two tablespoonsful *per diem* ought to be given, diluted with



FIG. 12.—Case of scurvy showing bending of the ribs.

water and well sweetened with sugar. If the bowels tend to be a little loose, this is no contra-indication. Tinned tomato juice may be used if none of the above mentioned fruits is available. Potato cream suggested by Dr. Still made by adding the floury portion of the potato immediately beneath the skin to cow's milk, to the consistency of cream is most useful. Six to eight teaspoonsful of this should be given during the day. For children who cannot manage pure orange juice without vomiting or loose stools, Government orange juice which has been concentrated is suitable, or Vitamin C (ascorbic acid)—one tablet—about 200 mgm daily. Clinically concentrated orange juice or tomato juice, black currant purée or rose hip syrup will be found very much more effective than synthetic ascorbic acid, even when the latter is given in very large doses.

**Prophylaxis.**—All infants whether breast or bottle fed from a fortnight old should be given fruit juice daily, two to four teaspoonsful of orange or grapefruit juice, rose-hip syrup, black currant purée or tomato juice being given in a bottle diluted with water and well sweetened with

sugar. With such an antiscorbutic added it is immaterial what food is used as scurvy cannot develop. Where infants refuse or return fresh orange juice, tomato or grapefruit juice, one half to one teaspoonful, can be added directly to each bottle, immediately before the bottle is given.

### CELIAC DISEASE

Celiac disease may be described as a condition of chronic fat and carbohydrate malabsorption resulting in infantism.

**Incidence.**—Two girls are affected to each boy. Eighty per cent of Hardwick's<sup>1</sup> cases commenced between six and twenty one months. The greatest incidence is reached at about one year. At Great Ormond Street in the 15 years 1924 to 1938 the number of celiac cases was 78, out of a total number of new cases seen at the Hospital of 445,771, i.e. 0.016 per cent of new cases were celiacs. During the war period from September 1939 to 1942, i.e. 3½ years the total number of celiacs was 41 and the total number of new cases 41,000, i.e. an incidence of 0.008 per cent of celiacs. Celiac disease seems during the war years to be 6 times as common as in peace times.

**Clinical picture.**—The complaint is usually that the child is failing to thrive and lacks appetite. Marked infantism is a feature, a child of two or even three years weighing only 15 or 20 lb and being proportionately small. The abdomen is large and the rest of the body wasted (Fig 13).

On palpation of the abdomen the liver is usually not felt but faecal masses may be present. The buttocks are wasted and the child resembles a case of abdominal tuberculosis. The Mantoux and tuberculin patch tests, however, are negative.

**Stools.**—These are large, pale, offensive, and often putty coloured; they suggest an excess of fat present as soaps. Bouts of looseness may alternate with constipation.

**Fat metabolism.**—A biochemical examination of the stool will show that there is a marked excess of fat, but it is to be noted that the fat is properly split, or digested. A good example would be

Splitted fat	41.9 per cent
Unsplit fat	11.0
Total faecal fat	52.9

Of the normal dried stool, less than 30 per cent is fat, and of this fat approximately 75 per cent is split. If a case of celiac disease is put on a low fat diet, and despite this the total faecal fats remain high, this is important, as it suggests that the case is severe.

<sup>1</sup>C. H. Wickham, Infantile Celiac Disease, *Arch. Dis. Child.* Dec. 1939, xiv, 49.



Fig 13.—Celiac disease

**Carbohydrate metabolism**—The oral glucose tolerance curve is a flat one in cœliac disease. The child does not absorb carbohydrate properly from the bowel.

**Vitamin "A" Absorption Test**—A fasting blood specimen is withdrawn, after which 7 000 I U of vitamin A per kilo of body weight is given orally in the form e.g. of Haliverol. Crook's Halibut Liver Oil contains 70 000 I U of vitamin A per c.c. Specimens of blood are then withdrawn 2 hours and 5 hours after the administration.

The vitamin A content of the blood in a normal child rises to an average maximum of 130 units in three to seven hours. In both cœliac disease and fibrocystic disease of the pancreas the rise attains a maximum of only between 10 and 20 units in a similar time.

**Blood calcium**—This tends to be low and in some cases there is actual tetany.

**Blood phosphorus**—In the majority of cases this is low, usually below 1 mgm. per cent.

**Blood phosphatase**—This is not raised in cœliac disease.

**Gastric secretion**—In 11 out of 15 cases reported by Hardwick there was a hypochlorhydria and in the remaining four cases achlorhydria.

**Duodenal secretion**—Trypsin is present in normal quantities.

**Bones**—The majority show osteoporosis but a proportion show active rickets, especially when the disease is beginning to heal.

**Anæmia**—In 21 out of 29 cases of Hardwick's there was a hypochromic anæmia. In the remaining 8 cases the anæmia was orthochromic or hyperchromic. Smallwood thinks that in these latter the spleen is usually palpable and the anæmia is macrocytic.

**Ætiology and pathology**—Recent work by C. D. May and others suggests that cœliac disease is due to deficiency of some substance found in liver, probably one of the vitamin B complex factors. Certainly in a small series of cases treated recently at Great Ormond Street on a full normal diet, the stools became normal and the children improved in health after injections of liver and vitamin B complex (Lederle). In May's series\* the ability to absorb fat and carbohydrate returned to normal after this treatment. At autopsy on cases of true cœliac disease the bowel wall is thin and wasted, almost like tissue paper; the liver is small but otherwise normal and the appearance of the other organs is what would be expected in any severe wasting disease (Fig. 14).

**Prognosis, mortality**—In the series reported by Hardwick the mortality was 86 per cent. Other series showed a mortality from 6 to 50 per cent, and average about 20 per cent. Severe diarrhoea, with broncho-pneumonia, was a frequent termination. The majority of the deaths took place between one and three years of age.

**Course**—The disease is rare in adults, thus one must conclude that the majority of cases recover. Before puberty these children remain rather short with a tendency to be unduly fat, but after puberty they

\* L. C. Parsons, "The Bone Changes Occurring in Ixonal and Cœliac Infantile Anæmia and their Relation to Rickets," *Arch. for Child.* 1937, 12, 1.

\* C. D. May, J. P. McGarry and K. D. Blackfan, "Notes Concerning the Cause and Treatment of Cœliac Disease," *Journ. Ped.* Sept. 1919, No. 3, xxi, 292.

## CHAPTER VII

### DISEASES OF THE ALIMENTARY TRACT

**Composition of normal stools.**<sup>1</sup>—Normal breast fed infants at first pass stools consisting of meconium and debris from the intestinal tract. These are greenish brown and loose. After the fourth or fifth day, as the breast milk appears they become yellower. Such stools are often described as mustard coloured and their reaction is acid. In artificially fed infants, the colour largely depends on the type of food and the stools are loose frequent and may be formed or almost formed. If the proportion of protein in the feed is high they are more formed and tend to have a cheesy or offensive odour they are alkaline to litmus. Malted foods tend to produce a brown stool and butter milk mixtures an olive green stool with a characteristic odour.

**Appearance of abnormal stools.**—For the purposes of examination the stools should first be broken up and pressed out so that the interior may be seen as stools which have stood for some time occasionally have a bright green exterior with a normal yellow interior.

**Grey stools** are due to an excess of fat and are characteristic of the older child who is livery or bilious the fat covering the normal yellow green bile this may also be seen in colic disease.

**White stools** may be seen when a very large quantity of fat is present or the bile is deficient. In congenital obliteration of the bile-ducts the stools remain white.

**Black stools** are the result of the presence of bismuth, iron or charcoal or they may denote blood, when the stools may be termed tarry. In *melana neonatorum* such stools are characteristic.

**Green stools** suggest that there is an increase of peristalsis hurrying the normal green bile through the bowel before it can be oxidized into the yellow bile-pigment. In acute infections of the bowel with diarrhoea such stools are constantly found. Accompanying the partially digested food there is often mucus and occasionally there may be blood.

Green stools are also seen where the damage to the bowel is due to fatty acids from overfeeding on fat or carbohydrate. In such cases the buttocks are also burnt. The starvation stool is green merely because it consists almost entirely of bile it is seen in congenital pyloric stenosis.

**Unusual ingredients and their meaning.**—Stools may contain the following:

(1) **Blood** which often accompanies a formed motion, may be due to an anal fissure. It may also be present in *melana neonatorum* and in ulcerative colitis. Haemorrhoids and rectal polypi are not uncommon in children and may give rise to bleeding. The "redcurrant jelly" motion of intussusception is characteristic.

<sup>1</sup> Donald Paterson "Normal and Abnormal stools in Infants," *Maternity and Child Welfare* July, 1924, 1.

(2) *Mucus* suggests that there is catarrh of the bowel, when it appears on the outer surface of the stool only it denotes colitis rather than enteritis, as in the latter the mucus is intimately mixed with the stool. Much mucus is passed after a dose of castor oil which causes acute irritation of the bowel wall. Mucus is also quite common during teething.

(3) *Pus* is present when there has been inflammation of the bowel wall, such as is found in true typhoid dysentery infections.

(4) *Curds* are the most common abnormal constituents of the stool and are almost always composed of fat. They dissolve in ether and float on water. Curds are very seldom due to protein especially if the milk has been boiled. Protein curds have a characteristic appearance, being large, square and Lima bean like.

For worms which may appear in the stool, see p. 892.

**Reaction of stools.**—The stools of breast fed infants are normally acid, those of bottle fed infants are alkaline. When however, in the artificially fed infant, an excess of fat or sugar is present the stools may become markedly acid, owing to the presence of fatty acids.

## CONGENITAL ABNORMALITIES OF THE ALIMENTARY TRACT

During the years 1900 to 1925, 28 autopsies out of 6000 performed at the Hospital for Sick Children Great Ormond Street disclosed the presence of congenital abnormalities of the alimentary tract. Sheldon<sup>1</sup> analysed these as follows—

	<i>Atresia</i> 6	<i>Partial Stenosis</i> 2	<i>Lived</i> 9 days
Esophagus			
Duodenum —			
Above bile pap. illa	2	1	12 days
" "	2	1	
Below " "	2	—	
Small intestine	13	—	6 days
Large " "	3	2	
	28	6	
	==	==	

**Intestinal atresia.**—Occasionally an infant is born with complete atresia of the small intestine. When fed, the child vomits. The bowels do not move. If the atresia is high up it cannot be differentiated clinically from narrowing of the duodenum. At operation the true condition of affairs is seen. If the atresia is far down the small intestine, or in the large intestine, the abdomen is distended.

**Prognosis.**—Even if the bowel is actually patent (which it usually is not) its wall is thin and functionless. Operation is, as a rule, useless and death takes place within a week.

**Imperforate anus.**—This is a rare congenital abnormality. The anus may be completely absent, being represented by a dimple only. In such cases a surgical operation is undertaken by an incision in the centre of the perineum, and a search is made for the rectum, an artificial anus being made and kept open with bougies.

<sup>1</sup> W. Sheldon "Congenital Atresia of the Alimentary Tract," *Arch Dis Child*, 1926, 1, 2, 3



Sometimes the anus is present, but is shut off from the rectum by a septum. This must be incised and kept patent with bougies from time to time. When the malformation is extensive the outlook is not good.

### ESOPHAGEAL STRICTURE OR STENOSIS

The stenosis may be complete the œsophagus terminating in a blind end at the level of the bifurcation of the trachea. In a case of this kind vomiting occurs from birth and there is rapid wasting resulting in death. There may however be a communication between the trachea and the œsophagus so that anything taken passes into the lungs. Where there is merely a narrowing the child may live on for many years being under size and able to take thin fluids but no solids. Such cases are continually examined for cardiospasm (see p. 108) dilatation being attempted with bougies and mercury tubes, with or without anaesthesia. The author's experience is that such cases ultimately do very badly although with occasional dilatation they are improved.

When the stricture is congenital the site is almost invariably at the level of the bifurcation. When it is acquired from trauma with some corrosive, it may be higher up and the ultimate prognosis may be much better although there is a tendency to become progressively worse.

### CONGENITAL PYLORIC STENOSIS<sup>1</sup>

As the name indicates this condition exists at or before birth. The sphincter or muscle guarding the exit from the stomach is unusually thickened so that food cannot leave the pylorus. Symptoms of obstruction then occur.

**Sex-incidence.**—Pyloric stenosis is much more common in males than in females with a ratio of 6 or 7 males to each female.

**Age-incidence.**—The initial symptoms occur most commonly at the end of the second week after birth. Occasionally however, the vomiting commences at birth or as late as the tenth week. The condition is most common in the first children of young mothers.

**Pathology.**—The pylorus is enlarged mainly because of hypertrophy of the circular muscular fibres. On section, the mucous membrane is seen to be folded and tight and often a ball of it blocks the gastric end of the pyloric canal. There is much evidence that this is a congenital malformation as it has been discovered many times in premature and still born infants. The generally accepted theory of causation is that the hypertrophy is due to an inco-ordination between gastric and intestinal peristalsis. Recently, much evidence has been produced to show that pyloric stenosis is inherited and familial and ought to be looked upon as Mendelian recessive<sup>2</sup>. The tumour is firm, round or oval and the muscle-fibres on incision are hard and in some cases feel almost cartilaginous. Usually, there is associated gastritis. Microscopically, the circular muscle fibres of the pylorus are seen to be greatly hypertrophied and thickened. There is no doubt that the whole of the stoppage or stenosis cannot be accounted for by thickening of the pyloric ring, and it is the author's

<sup>1</sup>Grey and Pirie, *Lancet* 1912 II 515. Foryston, Ellis and Eryden, *Lancet* 1914, I, \*15. *Brit. Med. Jour.*, 1913 I 59. Petersen, *Congenital Pyloric Stenosis*, *Acta Med. Scand.*, Feb. 1912, 196.

<sup>2</sup>E. A. Cockayne and L. C. Petersen, "The Genetics of Congenital Pyloric Stenosis," *Ohio Jour. Sci.* No. 1, Jan. 1943, xliii, 1.

opinion that in all cases there is superadded spasm. The reason for this view is that these children undoubtedly do pass stools and when a certain degree of debility occurs death is usually due to diarrhoea.

**Symptoms and diagnosis.**—As a rule the infant is breast fed, but may be artificially fed and seems to thrive up to the age of two weeks, when projectile vomiting commences. The vomit is large and obviously



Fig. 15.—Congenital pyloric stenosis, showing peristaltic waves passing across the child's stomach.

to be success for a day or two, then the vomiting recommences. The diagnosis rests on —

- 1 The history of persistent large vomits
- 2 Obstinate constipation
- 3 Seeing vigorous gastric waves passing from left to right across the abdomen (Fig 15)
- 4 The palpation of a pyloric tumour

**Method of diagnosis**—When presented with a case of persistent vomiting with obstinate constipation pyloric obstruction should be suspected the child should be given a feed and gastric waves watched for. The mere standing out of the stomach is insufficient as this occurs in so called pylorospasm. Waves the size of a small tangerine orange may be seen passing from left to right across the abdomen. Very often two waves may be seen at the same time. The more wasted the child the better the peristalsis. Vigorous waves will be seen in every case of true pyloric stenosis if the infant is observed for any length of time. During the feed if the abdomen is palpated a pyloric tumour can almost always be felt sometimes superficial sometimes deep in the abdomen, immediately to the right of the right rectus high up and just beneath the liver margin. X rays after a barium meal may show a delay in the emptying time. In a doubtful case some help may be obtained from this procedure but the author has not found a barium meal helpful reliable or necessary, in the diagnosis of pyloric stenosis.

The only two conditions which in any way simulate pyloric stenosis are (1) congenital stoppage or stenosis of the duodenum and (2) pyloric spasm.

In congenital stoppage of the duodenum vomiting always begins at birth. Constipation is obstinate or complete. The vomit contains bile and is green. Gastric waves occur but no tumour can be felt. As many girls as boys are affected. X rays settle the diagnosis.

With pyloric spasm insufficient feeding or a tendency to swallow air is the most common symptom. The vomiting is intermittent and there may be remissions of several days constipation being similar to but much less persistent than that in stoppage of the duodenum, the child having normal stools or even small bouts of diarrhoea. Gastric waves are never vigorous.

**Treatment**—The diagnosis having been made the choice is between medical and surgical treatment. To make the diagnosis certain however may take several days and during this uncertain period gastric lavage relieves the child of the retained food and tends to prevent gastritis. Subcutaneous rectal or intravenous salines should be given if the child is markedly wasted.

**Medical treatment**—For a number of years this was carried out more or less successfully but with the introduction of operation it has largely fallen into disuse in this country. Nevertheless on the Continent especially in Scandinavia medical treatment appears to be very successful. The treatment which the author has found successful consists of gastric lavage carried out once or twice daily, with careful feeding. The feed

chosen is usually one, such as peptonized milk or breast milk, where the curd is extremely small and can pass through a small opening in the pylorus. One of the atropine preparations should be given 10 minutes before each feed, such as *Pylotropin* (Clay & Abraham Ltd. Liverpool), a dose of which contains  $\frac{1}{800}$  gram of *Eumydrin*. This should be slowly increased up to  $\frac{3}{800}$  gram, if necessary. Toxic symptoms are shown by erythema or hyperpyrexia. It may be necessary to keep up treatment for many weeks. The treatment has been used successfully in this country.<sup>1</sup> The *Eumydrin* should be given as lamellæ and placed on the infant's tongue, or dissolved in one or two drops of glycerin and administered in the same way. Fresh air and sunshine are useful adjuncts although the utmost care must be taken to prevent chilling the infant. In this country results from medical treatment even under the best circumstances are not good. To secure a medical cure demands the utmost care and attention and the most expert nursing. During these trying weeks a slight chill giving rise to pneumonia or a temporary relaxation of the pylorus with a short bout of diarrhoea may end the struggle unfavourably. The author prefers surgical treatment in all cases. Medical treatment is reserved for mild cases especially in female infants where the condition is not usually so severe, or in those infants who come under observation at the ninth or tenth week or later.

**Surgical treatment**—The essential for this is that the cases should be secured early. When the child is two three or four weeks old and especially when breast fed the mortality is practically nil. At the eighth or tenth week and when he is artificially fed the outlook is less hopeful. In preparation for the operation any dehydration must be made good. Saline and glucose are administered subcutaneously or intravenously depending on the degree of dehydration present. No atropine injection is given before operation but the stomach is washed clean with normal saline an hour before. The arms and legs of the infant should be enveloped in cotton wool, lightly banded on to prevent cooling, and when the child is on the operating table, the hands may be fastened to the pillow. Gas and oxygen is the best general anæsthetic. Recently, local anæsthesia has been used very successfully.

*Rammstedt's operation* consists of splitting the circular fibres of the pylorus so that they spring apart like the tyre on a wheel allowing the food to pass through. It is a short operation taking less than 10 minutes. The infant, when returned to its cot, must be kept very warm. For the next six hours its rectal temperature should be taken hourly, and if there is any hyperpyrexia bottles and covering should be removed and an ice bag placed on the head.

#### Post-operative feeding—

##### *First Day (Breast or Bottle Fed Infants)*

##### *Anæsthetic*

- (a) Local commence feeding *two hours* from time of operation
- (b) General commence feeding *four hours* from time of operation

<sup>1</sup> Helen Mackay *Proc Roy Soc Med (Sect Dis Child)* April, 1936 xxix 1\*10

*Feed* Half strength breast milk or half-strength humanized dried milk (see p. 401) for the first twelve hours from time of operation, as follows —

Local Anesthetic	Post Operatively under General Anesthetic	Amount of Feed
2 hours	4 hours	1 drachm
3	5 "	2 drachms
4	6 "	3 "
5	7 "	4 "
6	8 "	5 "
7	9 "	6 "
8	10 "	7 "
9	11 "	8 "
11	13	1 ounce
13	15	1 "
		<hr/> Total 6½ ounces

Where possible, the infant should be allowed to sleep from 12 midnight to 6 a.m. or at least for a few hours after completion of the above schedule.

#### *Second Day*

Breast fed infants may return to the breast (test feeds being done)

Bottle-fed infants should be offered full strength feeds

	Total Amount
2½ oz. two hourly from 6 a.m. until 6 p.m.	17½ oz.
2½ oz. 9 p.m. and 2½ oz. at 12 midnight	5 "
	<hr/> 22½ oz.

*Third Day* 3 oz. three hourly (seven feeds)

*Fourth Day* Normal feeds for the infant's expected weight

#### Post-operative treatment —

1 If post operative vomiting is persistent, give gastric lavage once or twice daily for 24 or 48 hours, according to the amount of residue withdrawn

2 Give Pylostromin (Lumydrin, grains  $\frac{1}{80}$ ) three hourly, 15 minutes before feeds

3 Repeat subcutaneous or intravenous saline if dehydration is marked

4 If bowels are not opened in 24 hours, give a glycerine suppository

**Prognosis of surgically treated cases.**—There are many series of 100 or more cases with little or no mortality. David Levi (1941) operated on 100 consecutive breast fed infants without a death. With good nursing and freedom from cross infection, there is extremely little mortality. It is of interest to see the fall in mortality at the Hospital for Sick Children over the years, as shown in the table opposite.

TABLE XIII

SUMMARY OF CASES OF PYLORIC STENOSIS TREATED SURGICALLY

Year	No. of Cases	Av Age on Admission (weeks)	Sex		Result	
			M	F	Cured	Died
1920	59	6.3	49	10	42	17
1927	46	6.4	44	2	37	9
1928	71	6.5	61	10	51	20
1929	55	6.8	43	12	43	12
1930	70	6.6	54	16	51	19
1931	52	6.5	47	5	31	21
1932	57	6.2	50	7	41	16
1933	53	5.9	45	10	42	13
1934	84	6.2	74	10	75	9
1935	56	5.7	50	6	50	6
1936	69	6.0	58	11	57	12
1937	75	5.8	63	12	48	7
1938	68	6.2	63	5	62	6
1939	92	5.7	82	10	86	6
1940	64	6.4	57	7	54	10
1941	51	6.1	43	8	47	4
1942	82	7.0	75	7	78	4
1943	57	5.8	47	10	57	0

(1st 6 months)

## DUODENAL STENOSIS AND ATRESIA

Atresia may be due to some developmental error in the gut itself associated with partial or complete absence of its lumen. Stenosis is commonly due to compression or constriction of the duodenum by abnormal peritoneal bands or adhesions; in this case the gut has a normal structure, but its lumen is partially or completely obliterated.

**Symptoms**—Vomiting is marked especially of bile stained fluid. Constipation is complete, and meconium only is obtained by washing out the lower bowel. Where the atresia is partial only, very small quantities of food may find their way through. The bowel is narrowed for a considerable length—sometimes throughout the whole of the small intestine.

The commonest form of duodenal stenosis is due to bands or adhesions. In such a case the clinical picture is extremely like that of congenital pyloric stenosis—there is marked vomiting with constipation, the infant fails to gain weight, and is restless and crying. The vomit contains bile as a rule, thus differing from that of pyloric stenosis. On examination, waves can be seen passing from left to right across the epigastrium, but they pass far out into the right flank. The pylorus cannot be felt in such cases. It may be necessary to give some bismuth to complete the diagnosis, when the duodenum is shown by X rays to be greatly dilated throughout.

**Prognosis and treatment.**—The outlook in true congenital atresia is extremely bad, for when the bowel ends blindly, or when there is a narrowing over any considerable length of bowel, the difficulties of the surgeon appear almost insurmountable.

The cases which react best to surgical treatment are those in which the stenosis is due to bands or adhesions and several recoveries have been reported. Gastro-enterostomy may be necessary where the bands cannot be freed. In every case a laparotomy should be performed and the child given the advantages of surgical treatment.

## VOLVULUS

This is extremely rare in childhood. It may occur at any age.

**Etiology**—It is due either to some congenital defect of the mesentery—mal rotation or hernia of the bowel through an incomplete mesentery—bands, or an acquired defect such as an adherent Meckel's diverticulum.

**Clinical picture**—The classical symptoms of obstruction are usually present, i.e., vomiting, pain, collapse. Occasionally there are recurrent cases which unfold

themselves and pass for bilious or acidous attacks. In such cases, however, the abdomen is full and distended rather than scaphoid as in a true bilious attack.

The treatment is surgical.

## THE MOUTH

### STOMATITIS

The simplest form of stomatitis is *thrush*, due to *Oidium albicans*, or *Monilia albicans*.

**Ætiology**—Thrush is an infection of the mucous membrane and submucous tissues by a vegetable fungus, the spores of which project from the surface and give the characteristic white appearance. It is commonest in infants who are debilitated and who become infected from contaminated dummies and teats.

**Clinical picture.**—The tongue, roof of the mouth and inside of the cheeks have a white patchy or furred appearance, as if the infant had just been drinking creamy milk, and thin clots had been left sticking to the mucous membrane. An attempt to wipe these off, however, shows the patch to be firmly adherent to the tongue or cheek. There is a tendency for the infection to work through the child so that it is accompanied by severe vomiting and diarrhoea. The infection may spread to the oesophagus and give rise to grave general symptoms, with blood in the stools. The author has recently had a case where this infection passed to the larynx with symptoms of respiratory obstruction.

**Treatment.**—Glycerin and borax or a simple mixture of chlorate of potash, water and glycerin applied to the mouth will clear up the condition in a few days. It is most efficacious, also, to wrap a piece of muslin about the finger and gently scrape the affected part until the patch is more or less removed. Spores broken off in this manner fail to reappear.

Thrush<sup>1</sup> may also be most efficiently treated by freshly prepared 1 per cent aqueous solution of gentian violet applied to the mouth twice daily for three days and once every day for two weeks.<sup>2</sup>

### HERPETIC STOMATITIS (APITHOUS STOMATITIS)

This has recently<sup>3</sup> been shown to be due to a virus infection and is often associated with herpes labialis. It shows a great tendency to recur. It may be very mild causing a slight temperature only with vesicles on the cheeks and tongue which rapidly give place to small raw or grey areas. There is usually some gingivitis. Very often however the infection is severe, accompanied by a high fever and much salivation with severe inflammation of the tongue, gums and cheeks. The child usually aged from one to three or older, is much upset, and may resemble a severe case of ulcerative stomatitis. There is loss of appetite, sleeplessness and general misery.

The administration of sulphadiazine appears to shorten the course somewhat, but the stomatitis takes a week or longer to subside. Local applications such as gentian violet do not seem to affect the course of this infection.

### ULCERATIVE STOMATITIS

This infection has also been called "trench mouth" and "Vincent's angina." It is impossible to say whether it is the lowered resistance of

<sup>1</sup> J. H. Liles, Oesophagi in Childhood. *Arch. Ped. Child.*, 1938, xiii, 211.

<sup>2</sup> H. K. Faber and Edith B. Clark, "Prevention and Treatment of Thrush," *Amer. Jour. Dis. Child.*, Sept. 1927, xxxiv, 498.

<sup>3</sup> T. F. M. Scott, A. J. Steigman and J. H. Convey, *Jour. Amer. Med. Assoc.* 1941, cxvii, 909.

the patient or the virulence of the infection which is the outstanding factor in the clinical picture

**Ætiology and pathology.**—The organisms are in most cases the spirochæte of Vincent and the fusiform bacillus. The condition is extremely infectious, and tends to affect institutions, asylums and schools, or groups of men in barracks. In families a mother or father with chronic pyorrhœa may be the source of infection. In private practice it is often the nanny who is at fault. Why the infection should at one time affect the tonsils only, as in an epidemic of ulcerative tonsillitis, and at another time the mouth only, as in an epidemic of ulcerative stomatitis, cannot readily be explained.

**Clinical picture.**—Some cases are extremely mild, showing only redness of the gums with swelling and œdema. This makes the teeth appear to sink back into the gums, just the tips being visible. In other cases ulcers appear at the base of the teeth and the portion of the cheek coming in contact with these ulcers also becomes infected. There may be sloughs or ulcers on the tongue.

The onset is quite sudden. The temperature rises in a few hours to 100° or 101°, the child becomes fretful, refuses its food and tends to dribble. In a certain proportion of cases the tonsils are affected, and in severe cases the saliva is fœtid. An infant is extremely ill. Profuse vomiting and diarrhœa may occur before the mouth can become seriously involved. Teething infants show the severest lesions, possibly because of the lowered vitality of the gums when the infection takes place.

**Treatment.**—In the mild form, chlorate of potash, 2 grains four hourly may be sufficient. Glycerin and borax, or glycerin and perchloride of iron applied hourly to the gums, may also clear up the infection. There is little doubt in the author's mind, however, that Bowman's mixture, which is as follows, is the most efficient method of treating this form of stomatitis:

R	Liq arsen	}	aa 120 minims
	Vin ipecac.		
	Glycer		
	Aq menth pip ad 1 oz		

For children of 2½ to 5 years of age, who will spit out the medicine, it can be applied directly to the gums and tongue by means of a soft camel hair brush, any excess being expectorated. For children who are too young to run this risk, however, 5 drops should be added to a tea-spoonful of water, and this should be applied to the tongue and gums 4 or 5 times daily by means of a piece of muslin wrapped round the little finger and dipped in the mixture. The action of the arsenic is to kill the spirochæte, and that of the ipecacuanha to kill the fusiform bacillus. The glycerin makes the mixture adhere to the affected parts and, since it is hygroscopic, it promotes a free flow of lymph. Other antiseptics have a reputation, and undoubtedly succeed, but probably none has such rapid and sure success as this mixture. The application of a 1 per cent solution of gentian violet is efficient in this condition also.

A recent paper by C. G. Parsons<sup>1</sup> summarizes 115 cases of stomatitis

<sup>1</sup>C. G. Parsons, *Arch. Dis. Child.* March 1910, 27, 51.



He noted that girls were more frequently affected than boys, and four fifths of the cases were under 3 years of age. It was commoner in artificially fed infants and those who sucked dummies and thrush was the commonest infection. In the older children Vincent's infection was commonest together with glandular enlargement. One per cent. gentian violet water solution was as good as any other form of treatment but alternatively he suggested tincture of merthiolate (Eli Lilly).

Care should be taken to see that the bowels are well open and for this purpose some mercury preparation such as grey powder, with magnesia in the morning is most suitable.

#### GEOGRAPHICAL TONGUE

This is a common condition and seems to have little significance. The tongue shows areas where the surface is pink and normal interspaced with areas which are slightly raised and greyish. The shape of the normal areas may be round or have a serraginous edge hence the name geographical tongue. The cause of the condition is not clearly understood but it is stated by some to be due to a low grade chronic stomatitis. Certainly it is not as a rule accompanied by ill health. It is stated by some authorities to be an accompaniment of the exudative diathesis.

**Treatment.**—There is no indication for treatment.

#### CARIES IN THE TEETH

Dental caries is extremely widespread. The teeth of the hospital class of patient are still gravely neglected.

**Evils of dental caries.**—A carious first dentition will undoubtedly lead to caries in the permanent teeth. This is brought about by the poisons of decomposed food and bacteria passing directly from the temporary tooth to the permanent tooth below. In addition when a permanent tooth appears in a mouth which contains carious teeth it is sure to become quickly infected.

A child with carious teeth is very much aware of its disability. It fails to chew on the side where the teeth are affected. The result is that food is swallowed in a poorly masticated condition and is consequently ill digested and ill absorbed leading to *malnutrition*. Swallowing and absorbing decayed food and bacterial debris which collects in the teeth overnight give rise to a dirty tongue and chronic gastritis. The author has rarely seen a perfectly healthy child with extensive dental caries.

**Nervousness** can undoubtedly be traced in some children to bad teeth possibly acting through lack of sleep and poor assimilation of food, the most highly strung nervous children seen at hospital and those most difficult to examine, have over and over again proved to have extensive dental caries.

Septic teeth infect the tonsils and give rise to *septic tonsils* with all the ills consequent on this condition. Septic teeth also cause *enlarged glands* in the neck, which in time tend to become *tuberculous*.

**Ætiology and pathology.**—Teeth become carious in the first place because of deficient or badly formed enamel. This is almost certainly the direct result of a rachitic diet low in vitamin D, badly balanced or short of calcium, and also of insufficient sunshine and fresh air. In addition,

\* M. Mellanby, "Diet and the Teeth," Reviewed in *Lancet* 1922, 1, 945. "The Influence of Diet on Caries in Children's Teeth," Med. Research Council Spec. Rep. Ser. No. 11 (H.M. Stationery Office London 1926) p. 12.

the child may inherit a family tendency to poor bone formation, and this may be an aggravating factor in the production of poor enamel

A second factor is a failure to keep the teeth clean and in good condition, a duty which obviously devolves upon the parent

Undoubtedly, severe illnesses tend to leave permanent defects in the teeth. A great portion of the enamel of the teeth is laid down while the child is in utero, and therefore the food of the mother and her general health at that time may be of the utmost importance

Another factor in the production of caries is an impaired saliva. The saliva normally has bactericidal properties, and anything which impairs this allows the bacterial flora of the mouth to flourish and attack the teeth

Finally, if children are given soft and pappy meals with sugar in them and if sugar is given between meals, the proper jaw movements and the friction of hard food on the molars are prevented and at the same time bacteria are allowed to flourish in the retained sugar lying in the interdental spaces. It sometimes happens especially in girls at puberty that a hitherto perfect set of teeth will suddenly show marked caries and an examination of the blood calcium and phosphorus in such cases will show a reduction of one or both, with a blood phosphatase much raised. Benefit is derived from calcium diphosphate, say 15 or 20 grains three times daily together with vitamin D, often best given in capsule form at this age and some artificial sunlight

**Care of the teeth.**<sup>1</sup>—Every child should have the teeth cleaned by scrubbing with a soft, boiled toothbrush twice daily from the age of 9 or 10 months. An alkaline mouth wash, such as milk of magnesia is sufficient, and as the child grows older the brush should be stiffer. Hard foodstuffs, such as rusks, crusts and crisp toast should form part of each meal, so that the gums are stimulated and the saliva is forced throughout the mouth. If the smallest hole appears in either the first or second set of teeth, a dentist should be consulted, the hole cleared out and stopped. No amount of trouble is too great to preserve the first as well as the second set of teeth as long as possible. The removal of the first dentition for caries is most detrimental as the jaw fails to expand if the first teeth are removed early, and the result is overcrowding of the second dentition. This removal, however, may be necessary where sepsis is present. No sweet food should be eaten before going to bed. The teeth must be cleaned the last thing after all food is taken. Cod or halibut liver oil is an essential in the diet of every child in this climate

## CONSTIPATION IN INFANCY AND CHILDHOOD<sup>2</sup>

**Constipation in the breast-fed infant.**—This is extremely common and among the causes are

1. Atonicity of the abdominal walls due to the child being *overweight* and *soft and flabby*. The condition can be improved by underfeeding the child and giving massage and sunlight

2. An *insufficient intake of fluid*, especially during the summer months

<sup>1</sup> A. T. Pitt. "The Treatment of Children's Teeth," *Public Health*, June 1937 II 495

<sup>2</sup> Donald Paterson. "Constipation and Its Management in Infancy and Childhood," *Brit Med Jour.*, Feb. 13 1936, I, 61

In addition to the breast, the child should be offered 4 to 6 oz of water daily, either from a bottle or with a teaspoon from a cup

3 *Faulty training*—The training to pass motions into a chamber should begin in earliest infancy, as described on p 11

4 If the child has an anal fissure he fears defecation A little ung hamamelidis (witch hazel) should be pressed into the anus both before and after a motion, and the motion kept soft with milk of magnesia Some infants have been so frightened by the pain that they still refuse to defecate, and it may be necessary to insert an infant sized glycerine suppository, each morning for several days, applying plenty of witch hazel ointment

5 A certain number of children suffer from *congenital anal stenosis*, that is the anus is actually too small and requires careful slow dilatation This is best done by inserting a vaselined little finger on several occasions

A little Sempolin or Petrolagar Emulsion, with or without phenol phthalein is a great help if given occasionally only For the child who requires a slight aperient milk of magnesia (Phillips's) will be found excellent if given before the first breast feed in the morning

6 In the *starved or underfed infant* constipation is often one of the first signs of failure of the milk supply

**Constipation in the artificially-fed infant.**—The above remarks are also applicable in great measure to the artificially fed infant, but in addition some other points must be stressed

All infants fed on cow's milk are slightly more constipated than the normal breast fed infant The higher the proportion of protein or curd in the diet, the more alkaline and constipated the stool The reverse is true that the more fat and sugar present and the higher the acid content, the more likely the child is to have diarrhoea and sore buttocks A nice balance must therefore be aimed at between the constipating protein and the acid, diarrhoea producing fat and sugar

When an infant is inclined to be constipated and the sugar and fat-content are not unduly high a slight increase is indicated Mellin's Food replacing lactose or Demerara sugar, will be found distinctly more relaxing Olive oil extra cream, and especially castor oil, should be condemned in the treatment of constipation For the occasional attack of constipation or formed motions a teaspoonful of Phillips's milk of magnesia given first thing in the morning should be sufficient, and for the older infant  $\frac{1}{2}$  to 1 grain of grey powder given at bedtime, with the magnesia the following morning, is excellent

Pulped stewed apple or prune or fig juice, may be useful as an addition to the diet In older infants cereals of the coarse variety, such as rusks, crisp toast and wholemeal bread, and such vegetables as sieved spinach, sprouts, greens and cabbage all help to give the motions bulk and prevent constipation Where the child is flabby and soft, slight under feeding, with massage, fresh air and sunshine, tend to strengthen the abdominal muscles

The psychological aspect should be considered, even in the infant It is necessary to attend to the bowel movements with machine-like regularity He must not be allowed to think that the failure of his bowels to move is of the utmost importance A studied air of unconcern is to be preferred,

but the situation should be handled with tact and firmness, making it plain what the child is on the chamber for, and what is expected of him

**Constipation in the older child.**—Constipation in the older child has various causes

1 The mother or nurse may have failed to establish regular habits of evacuation in infancy. Insufficient importance has been attached to the emptying of the bowel or insufficient time devoted to this act, with the result that it is put off and neglected with subsequent constipation. To correct this state of affairs, regular habits must be established, the child "sitting down" at the same time daily and, if unsuccessful, repeating the process later in the day

2 Atonicity of the bowels as part of a general debility may be found after a debilitating illness or when the child is in bed. The bowel and abdominal wall become soft and flabby, so that the fecal contents cannot be pushed forward. This is also seen in flabby soft children especially those with large distended abdomens who suffer from stercor dyspepsia. The 'nervous exhaustion child' or nervous dyspeptic also comes in this class. The muscles are hypotonic and very little intra abdominal pressure can be exerted on the intestinal contents

Until the general condition is improved, no improvement in muscle-tone is likely. This may mean changing the child's whole life and regime. A change to the seaside, general tonics, fresh air, real or artificial sunlight massage, adjustment of the diet are all indicated

3 Anal fissure or hæmorrhoids occur in older children. In all cases where pain is complained of, a careful examination should be made and gall and opium ointment or ung. hamamelidis (witch hazel ointment) should be applied both before and after defæcation

4 The normal child who is getting insufficient food, or food which is too completely absorbed, will tend to be constipated. The diet should contain a certain amount of residue producing food. Such is to be found in whole meal bread and cereals of all kinds and especially in vegetables and fruit and a liberal supply of each of the last two should be present in every child's diet. It is seldom that green vegetables or raw or cooked fruit are given too freely, the great tendency being to underfeed children on these articles of diet. Bemax and Kellogg's All bran are also useful

On the other hand, the diet may be liberal but wrongly balanced. A diet containing too much protein will undoubtedly tend to produce constipation, as protein gives rise to alkaline motions which decrease peristalsis

Careful review of the child's food with these points in mind will often reveal the error

5 The diet of many children is deficient in fluids, especially in summer time. In this way the motions become dry and crumbly. Drinks of water should be given on waking and during warm weather, even between meals, and every child should drink from one pint to a pint and a half of fluid daily as a minimum. Quantities less than this will tend to produce a dry intestinal content

6 In the older child at school it is essential that sufficient time be given for proper attention to the bowels. No day should elapse without a motion,

and the child should be trained to report his failure at once, so that the proper steps can be taken to ensure one the following morning

**Medicinal treatment**—In older children *liquid paraffin emulsion* or even plain liquid paraffin should be relied upon. Such preparations as *Petrolagar Semprolin* and *Agarol* especially when combined with phenol phthalein are most efficient provided they are given not more than once or twice weekly. Some Eno's Fruit Salts or syrup of senna, last thing at night or first thing in the morning should keep the most obstinate bowel in order. *Castor oil* should be avoided in all cases of constipation, as it tends to leave the child more constipated than before and should be reserved for children who are inclined to diarrhoea. *Senna pods* may be given daily or small doses of *cascara* as a bowel tonic. *Taxol*, one to two tablets daily will be found extremely satisfactory. Vegetable laxative tablets, prepared by various firms are useful as an occasional aperient. *Salutaræ* and other forms of saline are best given at least an hour before breakfast.

Some old Great Ormond Street favourites are

*Mist Rhei cum Soda*

Rhubarb in powder 1 grain  
Sodium bicarbonate 2 grains  
Syrup of ginger 6 minims  
Peppermint-water to 60 minims

*Mist Aloes Co*

Tincture of nux vomica 1 minim  
Tincture of ginger 2 minims  
Liquid extract of hyoscyamus,  $\frac{1}{2}$  minim  
Aloes  $\frac{1}{2}$  grain  
Syrup of senna 15 minims  
Dill water to 60 minims

*Mist Cascara*

Liquid extract of cascara	} of each 15 minims
Liquid extract of liquorice	
Syrup of orange peel	
Chloroform water	

*Mist Cascara cum Malto*

Liquid extract of cascara 10 minims  
Extract of malt to 60 minims

*Syrup Ficorum Laxans*

Fluid extract of senna-pods 6 minims  
Fluid extract of cascara 3 minims  
Compound tincture of rhubarb, 3 minims  
Syrup of figs to 60 minims

## DISTURBANCES OF DIGESTION

### SUMMER DIARRHŒA

#### FEBILE DIARRHŒA GASTRO ENTERITIS

**Classification of diarrhœa.** 1 Infective diarrhœa (due to pathogenic organisms, such as those of Sonne, Flexner, Shiga and Y Dysentery and probably undescribed virus infections)

2 Dietetic diarrhœa (due to over feeding, particularly on fat and carbohydrate)

3 **Symptomatic diarrhœa** (i.e. diarrhœa secondary to some parenteral infection such as otitis media, tonsillitis, pyelitis or pneumonia, which causes an accompanying intestinal upset)

The proportion of each group among the cases of diarrhœa in any summer in any country, will depend upon the climate and economic conditions. Thus in America and Australia, where extremes of heat are found, infective diarrhœa appears to be relatively common, but is a rarity in the British Isles.

TABLE XIV

INFANT MORTALITY (ENGLAND AND WALES) DISTINGUISHING MORTALITY FROM  
DIARRHOEAL DISEASES

(Deaths under one year of age per 1 000 live births)

Year	Diarrhoeal Diseases	Other Causes	All Causes
1861-65	15	136	151
1921-25	8	68	76
1930	5	50	60
1937	5	52	57
1939	4	46	50
1941	5	54	59
1942	4	45	49

From the above table it is obvious that diarrhoeal diseases have a more or less stationary mortality

1 **INFECTIVE DIARRHŒA** (epidemic gastro enteritis).—**Ætiology and pathology.**—This occurs more frequently in summer time. Blood and mucus are characteristically present in the stools, but *post mortem* little or no ulceration of the small or large bowel is found. The liver is fatty, there is cloudy swelling of the kidneys and enlargement of the mesenteric glands. A terminal broncho pneumonia is common.

**Clinical picture.**—A previously perfectly healthy child suddenly develops acute diarrhœa and vomiting, and dehydration is rapid: the skin becoming inelastic, the tongue furred and the fontanelle depressed: the stools finally consist of mucus only. There is a low blood pressure and there are symptoms of general intoxication. (For prognosis and treatment, see p 104.)

2 **DIETETIC DIARRHŒA**.—The Infant Welfare Movement with education in infant care and feeding is largely responsible for the relative rarity of this type.

**Ætiology and pathology.**—Badly balanced feeds, such as those with a very high protein or high fat or unduly high carbohydrate content are responsible, if an over zealous mother presses such a feed on her infant, particularly in warm weather. Often the infant is not being offered drinks of water between feeds, and must take unnecessary food, merely to obtain fluid and quench its thirst. Exactly what happens inside the alimentary tract is not known, but it is assumed that fermentation or indigestion occurs. *Post mortem* no lesions are visible in the alimentary tract.

**Clinical picture.**—Unlike the previous group, the onset is slow and insidious. There is at first a failure to gain in weight and a tendency to refuse food, with finally a slight rise in temperature. The infant is not very ill in the initial stages but, unless the error in diet is corrected and

the management improved, it passes rapidly into a state of dehydration. At first curds or frothy motions are passed, later, with increased peristalsis the stools become bright green or resemble chopped spinach, burning the infant's buttocks. The child does not vomit, although it may begin to in the later stages. (For prognosis and treatment, see below.)

**3 SYMPTOMATIC DIARRHŒA (Parenteral infections) *Ætiology and pathology.***—By far the commonest cause of severe diarrhœa and vomiting or gastro-enteritis is a naso-pharyngitis, usually not accompanied by nasal discharge. Often there is otitis media, or a mastoid—quite unsuspected. Pyelitis may show itself only as gastro-enteritis. Too often the attention of the mother and doctor is diverted by the diarrhœa, and the real cause is overlooked. Why an infection in some other part of the body should produce a gastro-enteritis is not clearly understood. Couper summarised a number of cases of severe diarrhœa complicated by otitis media and mastoiditis and found the Schilling hæmogram of the greatest use in determining which cases should be operated on and which should not. Examination of the white cells of the blood, and particularly of the granular series showed an abnormality in the polymorphonuclear leucocytes. It is suggested that in a normal infant about 7 polymorphonuclears in 100 are primitive or immature cells, the remainder being mature multi-lobed polymorphonuclears. With infection of the middle ear or mastoid there is a shift to the more immature type and as high as 80 or 40 per cent. of the cells may be immature. Many observers support Couper's views.

It is obvious therefore, that a thorough and exhaustive clinical examination including the ears and urine should be made in every case of gastro-enteritis.

### **Prognosis and Treatment in all types of Gastro-Enteritis.—**

**1 INFECTIVE DIARRHŒA**—There is a polyvalent anti-dysenteric serum for the cases in which a dysenteric organism has been isolated. With the advent of the Sulpha drugs the treatment of choice is sulphaguanidine, the dose being from one to 2 grains per lb. body weight per day for three or four days. In *Sonne dysentery* there is a much less severe clinical picture although occasionally this infection proves fatal in very small infants. The disease runs a short course of fever from 3 to 5 days, with blood and mucus in the stools and severe vomiting. Sulphaguanidine appears to be specific in most cases.

(For treatment of dehydration see p. 105.)

**2 DIETETIC DIARRHŒA**—In slight and early cases, castor oil, one to one-and-a-half teaspoonsful, should be given, and all food stopped for 24 hours the infant being given bland fluid, such as plain water or sugar water. At the end of 24 hours' starvation, without waiting for the stools to become yellow, smooth and normal, the child should be fed on a freshly adjusted feed.

**3 SYMPTOMATIC DIARRHŒA**—Any pyelitis should be treated (see p. 202). If the ear-drums are red and bulging or the pharynx inflamed or bronchitis present, suitable treatment, such as one of the Sulpha drugs,

should be instituted. In other words, the underlying infection must be treated.

### DEHYDRATION AND FEEDING

The most important symptom to be treated in cases of gastro enteritis is dehydration. There are several alternative methods —

1 *By mouth* — Give bland fluids such as water, sugar water or fruit juice and water. For more serious cases give Hartmann's solution (see prescription below) or half normal saline with 5 per cent glucose. 2½ ozs per lb body weight per day plus extra for dehydration at two to three hourly intervals. This method is not suitable where dehydration is very marked or the infant is vomiting.

2 *Subcutaneously* — This is not a practical method for introducing more than a very small quantity.

3 *Intra peritoneally* — This method is not a liked as the fluids tend to become infected in debilitated infant.

4 *Intravenously* — The internal saphenous or veins in the cubital fossa or the veins of the scalp are the best. A Biteman's needle (Allen & Hanbury) should be used and half strength Hartmann's solution with 5 per cent glucose. The vacoeters should be calibrated in 10 c.c. units by marking a strip of adhesive strapping the length of the vacoeter. This allows for adjustment of the drip, so that from ½ to 1½ oz per hour of the Hartmann's solution can be given steadily day and night. Records should be made hourly of the quantity given. 2½ oz of fluid per lb body weight per day should be administered.

### SCHEDULES OF TREATMENT

The following is a method used for moderate and severe cases of gastro enteritis at the Hospital for Sick Children, Great Ormond Street, details being adjusted to suit individual cases. The type of case for which it is reserved are those in which —

(a) the stools are frequent, watery, offensive and contain blood and/or mucus,

(b) the child vomits persistently,

(c) there is a rise of temperature,

(d) the child looks ill and dehydrated.

1 Castor oil under 1 year give 1 drachm over 1 year give 2 drachms.

2 Stop all milk and give, 2-3 hourly by mouth half strength Hartmann's Ringer lactate solution. Add a little saccharine if necessary.

3 *Daily total fluid intake* — 2½ ozs per lb body weight, plus extra for dehydration, not exceeding 10 ozs. Slight dehydration may be restored by subcutaneous saline, given early.

4 *If dehydration is marked put on intravenous drip* of half strength Hartmann's solution containing 5 per cent glucose for 24 hours, with only sips of water by mouth.

Weight of Infant	Amount per 10 oz	Total in 24 hours
5 lbs.	15 c.c. (½ oz.)	360 c.c. (12 oz.)
7 lbs.	20 c.c. (⅔ oz.)	480 c.c. (16 oz.)
10 lbs.	30 c.c. (1 oz.)	720 c.c. (24 oz.)
15 lbs. or over	45 c.c. (1½ oz.)	1080 c.c. (36 oz.)

After 24 hours, feeds should be introduced according to pyloric schedule.



(Note 6—second method) The drip must be slowed provided dehydration is corrected but must deliver enough to make up the day's total to the required amount. As the feed is increased so the drip is reduced. It should be the aim of treatment to make the baby independent of the drip by the end of the third day.

When intravenous therapy has to be employed over a prolonged period the replacement of lost plasma proteins may be advisable. This is best done by using equal parts of half strength Hartmann's lactate solution and human serum thus obviating the technical difficulty of getting the rather sticky full strength fluid to run through a slow drip.<sup>1</sup>

#### 5 Choice of milk feed —

- (1) Breast milk if obtainable
- (2) Half cream dried milk with or without lactic acid (e.g. half cream Lactac Co. and Gate Ltd.)
- (3) Baled milk with or without lactic acid (babies over 6 months dislike Lactic acid milk)
- (4) Unsweetened condensed milk

6 Method of introducing milk feed — (a) First method (When on Hartmann's solution by mouth) After 24 hours continue the feeds, replacing part of the Hartmann's solution by the milk chosen, as follows —

1 part milk to 3 parts Hartmann's for 12 hours	
parts	parts
4	12
5	12
	12
	12

and so on until the correct milk strength for age is reached

Sugar should be gradually introduced when a half strength milk mixture is reached. (N.B. Mix the Hartmann's solution and milk immediately before the feed otherwise it tends to curdle.)

Dried or condensed milk should be reconstituted to whole milk before adding the requisite part to the Hartmann's solution.

(b) Second method — (Pyloric schedule if feeding to be used when changing from intravenous drip to bottle feed) Choose one of the milk feeds as in No. 5, and dilute as described in No. 6.

Drachms		No. of Feeds	Total No. of hours	Total No. of oz.
1	hourly	3	12	3½
2		3		
3		3		
4		3		
1	1½ hourly	4	12	6
Ounces				
1		3		
1½		6		
2	2	6	24	21
2½		6		
3		6		
3		6		

It may be necessary to go more slowly than this, and even to alternate milk feeds with Hartmann's solution. For large babies the quantities may be doubled. Baby should have absolute rest, if possible, from mid night to 6 a.m.

7 *A stomach wash-out with normal saline, given early in the treatment and occasionally repeated but not more than once per day is often effective in checking the vomiting. A rectal wash out with normal saline, given early in the treatment is effective when stools are particularly offensive with much mucus.*

8 *Drugs*—For collapse coramine 0.5 c.c. intramuscularly every 2 hours up to 6 injections.

9 *Fresh air*—An over heated room, lacking ventilation increases a baby's disposition to vomiting and diarrhœa. Warmth by hot water bottles may be necessary but open air ventilation is essential.

10 *Special note*—There is a tendency to over treat these babies. They collapse very easily. All medical and nursing procedure should be reduced to a minimum.

#### HARTMAN'S SOLUTION

(Liquor Ringer Lactatis) (Supplement to B.P.C. 1934 Part III Formulary 1941)

	Metric	Imperial
Lactic acid	2.4 ml.	73 minims
Solution of sodium hydroxide	1 sufficient	quantity to neutralize the lactic acid
Sodium chloride	6.0 gm.	5½ grains
Potassium chloride	0.4 gm.	3½ grains
Calcium chloride	0.2 gm.	1½ grains
Distilled water to	1 000.0 ml.	90 fl. oz.

#### APPLE TREATMENT

Success has recently been claimed for the apple treatment in cases of severe diarrhœa in older infants and young children. The apples are grated finely, and freshly prepared at each feed. From one teaspoonful to a whole apple is given at each feed according to age. Between feeds the fluids are made up to 2½ oz. per lb. of body weight per day by giving weak, freshly brewed tea. Nothing apart from the apple and tea is given for three or four days.

#### Prognosis—Infants under 2 years

- 1 The mortality is about 25 per cent.
- 2 The more the infant is under its expected weight the higher the mortality.
- 3 The mortality is highest in the first six months of life.
- 4 Debilitated infants contracting the disease in hospital show a much higher mortality than healthy infants contracting it at home.
- 5 The earlier the treatment the lower the mortality.
- 6 *The more dehydrated the infant the higher the mortality.*
- 7 Severe vomiting and diarrhœa for more than one week carry a bad prognosis.

Over the age of 2 years the mortality is for practical purposes nil.

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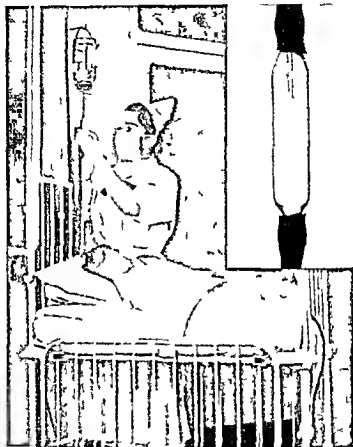


Fig 16 Nurse adjusting the continuous intravenous drip which is being given into a vein in the ankle

#### CARDIOSPASM (ESOPHAGECTASIA)

This is a very rare condition in infants and young children and only with difficulty distinguished from true oesophageal structure due to some congenital malformation or acquired by trauma

**Symptoms**—The child is, as a rule, under nourished and under sized but occasionally looks normal. Once or more often in the day it vomits, especially at any attempt to

<sup>1</sup> Donald Pearson: A Case of Esophagectasia in a Child. *Brit Jour Child Dis* January March 1921 XVII, 2

Wilfred Shelton and A. G. Oliver: Obstruction of the Oesophagus in Childhood. *Arch. Dis. Child* 1920, IV 247

take solid. Fluids are better retained. A bismuth meal may pass rapidly into the stomach but as a rule shows a definite narrowing. The site of true cardio-spasm is most commonly at the extreme lower end of the œsophagus where there is a dilatation or pouching.

**Ætiology**—Whether there is a primary ulcer with resulting spasm of the muscle due to a stimulus of the nervous control, as Cameron states<sup>1</sup> it is impossible to say. Certainly the condition is intermittent and there are brief periods when the spasm is present and vomiting is frequent then periods sometimes of many months when the child appears normal. The condition may persist into adult life.

**Treatment**—As in adults, treatment is usually successful. Dilatation of the narrowed portion of the œsophagus by means of a rubber tube filled with mercury is indicated. In a recent case aged eight under the author's care at the Westminster Hospital the child gained several pounds and his vomiting completely ceased when a mercury tube was passed before meals. He learned to pass it himself and he was sent home with the tube which he continued to use.

## INFLAMMATORY STATES APPENDICITIS<sup>2</sup>

This is the most common and may be the most serious of all the inflammatory abdominal conditions. Early diagnosis is of the utmost importance.

**Ætiology and pathology**—Appendicitis may occur at any age but is uncommon before three. There is no sex predominance. By some it is claimed to be due to an infection gaining entrance to the blood stream possibly through the mucous membrane of the bowel or through the tonsils and passing directly to the appendix. Others contend that it is a local abscess starting in the lymphoid tissue of the appendix due to linking or blocking of the appendicular lumen. Certainly threadworms filling the appendix have been known to give rise to a typical attack. An association between appendicitis and rheumatism was pointed out many years ago by Poynton and Paine.<sup>3</sup> There is ground for believing that in some cases there may be a specific infection. Epidemics sometimes occur in schools and institutions. Once the organ has become inflamed it may be distended and congested. This inflammation may extend through the walls to the peritoneal coat and sets up local or general peritonitis. In a very virulent or severe infection local gangrene may develop with perforation the faecal contents of the appendix finding their way directly into the abdomen. Where the omentum becomes adherent in the region of the appendix, the whole may be sloughed off and an abscess may form.

**Symptoms and diagnosis**—The chief symptoms are abdominal pain accompanied by vomiting with tenderness and rigidity of the lower right rectus (*McBurney's point*). The clinical picture varies with the site of the appendix and the stage of the disease. When the appendix is retrocaecal the clinical picture is often atypical pain and tenderness being much less marked than in the average case, and vomiting absent. Often the symptoms are so slight that they are taken for an ordinary bilious attack. Fever is not constant, but is usually present, especially in the smaller

<sup>1</sup> J. A. M. Cameron, "Œsophageal spasm in a Child," *Arch. Dis. Child.* 1927, lii 3-3.

<sup>2</sup> I. F. Barrington Ward, "The Abdominal Surgery of Children" (Oxford Medical Publications) p. 121.

<sup>3</sup> and H. Nixon, "Acute Appendicitis in Children," *Am. Jour. Dis. Child.* No. 6 June 1935 li, 149.

<sup>4</sup> B. W. Williams and R. H. Rogers, "The Mechanism of Appendicitis," *Lancet* Jan. 6 1934 li, 9.

<sup>5</sup> Poynton and Paine, *Lancet* 1911 li 129.

children. In every case of doubt a rectal examination should be made. In doubtful cases the very greatest importance should be attached to the time of onset of the pain. Pain which precedes vomiting is significant, pain which follows on vomiting especially if much retching has occurred, is not of so much clinical importance. Tenderness and rigidity or spasm of the abdominal muscles coupled with abdominal pain make the diagnosis complete.

The appearance of the patient is also important, the eyes being sunken and the tongue furred and dry. The pulse is raised out of proportion to the degree of fever and is bounding. As a rule, constipation is obstinate, but occasionally there may be a tendency to small infrequent motions.

**Differential diagnosis.**—Usually the diagnosis is easily made, but occasionally appendicitis may be mistaken for a *bilious attack*. In such cases the absence of abdominal pain with tenderness and rigidity, should be of some help. In the retrocaecal type however, there is a close similarity between the two and a rectal examination alone will decide the diagnosis. *Migraine in childhood* with marked abdominal symptoms may simulate appendicitis. *Diaphragmatic pleurisy* and *right sided pneumonia* may present a similar picture in the early stages. The general appearance of the child should be of help, as the face is flushed and the respirations are increased. A rectal examination does not reveal tenderness.

*Inguinal and mesenteric adenitis* when the deep group of glands in the inguinal region on the right side become inflamed, may be difficult to distinguish from appendicitis. The general system is upset is less marked as a rule and colicky pains tend to pass off rapidly. Fortunately, this condition is rare. *Pyelitis* in the right kidney may simulate appendicitis closely but an examination of the urine should settle this point. In *general peritonitis due to the pneumococcus* tenderness is present throughout the whole abdomen, toxæmia is extreme, and pain has not been limited at any time to the right iliac fossa. Only by local examination and consideration of the child as a whole can a differential diagnosis be made. Inserting a needle into the peritoneal cavity has been suggested by some observers to decide the diagnosis. *Acute follicular tonsillitis* is sometimes accompanied by colicky pains about the umbilicus (*umbilical colic*). The mode of production of these pains has not been satisfactorily explained, but they are universally recognized. With the subsidence of the tonsillitis the abdominal pain disappears.

**Prognosis.**—The sooner the diagnosis is made and treatment instituted the better the prognosis. Once general peritonitis has supervened, the mortality rises very rapidly especially in children under the age of six. With early diagnosis and efficient surgical treatment the mortality should be nil. In 467 consecutive cases Bruce<sup>1</sup> at the Children's Hospital, Aberdeen had a mortality of 1.9 per cent. Herzfeld,<sup>2</sup> at the Royal Edinburgh Hospital for Sick Children found the average duration of illness before admission was two to three days. Out of 100 consecutive

<sup>1</sup> G. Bruce. Diagnosis and Treatment of Acute Appendicitis in Children. A Review of 467 Consecutive Cases. *Lancet* 1911, i, 174.

<sup>2</sup> G. Herzfeld. *Edin Med Jour.*, Dec. 1922, x li, 59.

cases of appendicitis, 84 had been given operations and of these 27 required drainage. In only 23 per cent was the appendix in the classical position at operation. In the majority it was retrocaecal in the pelvis or high. There were 12 deaths out of 493 cases, half of them in children under the age of three years.

*Treatment.*—*The treatment is purely surgical.*

### CHRONIC OR RECURRENT APPENDICITIS

The question whether true chronic appendicitis occurs in childhood is debatable. Probably, the term *recurrent appendicitis* is much nearer the truth, but even recurrent appendicitis must be considered rare.

**Symptoms and clinical picture.**—After a mild or subacute attack of appendicitis, with fever, pain, vomiting and tenderness, the symptoms may subside and the whole illness may be regarded as merely a bilious attack. Some weeks or even months later there may be a recurrence, this time possibly with sharp stabbing pains or nausea, coated tongue, lassitude and a low, 'grumbling' temperature. The temperature may never have properly settled between the first attack and the second, although on examination between the exacerbations there is little or nothing to be found. Such recurrences may go on for some months or even years before the child is seen in one of the attacks and a diagnosis is made.

**Differential diagnosis.**—Some of the conditions which may simulate recurrent appendicitis are inflamed abdominal glands, tuberculous or otherwise, intermittent hydronephrosis, calculus, adherent Meckel's diverticulum, a partial volvulus, chronic intussusception, follicular tonsillitis with so-called umbilical colic or acidosis attacks. It is only by keeping such conditions as these in mind and excluding each when possible that a correct diagnosis can be made.

In a series of suspected cases submitted for X-ray examination no very real help was obtained. The barium certainly passed readily into the majority of normal appendices, but where it did not no definite evidence of disease was necessarily found at operation.

Some authorities regard so-called aculosis or cyclical vomiting attacks as merely recurrent appendicular attacks and remove the appendix as a routine. Unfortunately although occasionally the appendix is the source of the trouble its removal does not always cure and the author has known patients complaining of the same symptoms after operation.

Very often the question of appendicitis is raised on account of abdominal pain, and this gives rise to constant fear in the mother's mind that it may recur unnoticed. In these circumstances although the diagnosis may not always be absolutely clear, the removal of the appendix appears to be justified in order to set the mother's mind at rest.

In true acute appendicitis, where there is acute inflammation and later abscess formation, there is seldom, if ever, a history of previous appendicitis attacks. There seems to be little evidence, therefore, for believing that true chronic appendicitis does exist in childhood. Recurrent appendicitis or—to use the much better term—subacute appendicitis should be diagnosable in the acute stages, which are the equivalent in childhood of adult appendicitis.

A continuous pyrexia, for which no explanation could be found, together with recurrent mild abdominal pain, would, even in the absence of physical signs, suggest recurrent appendicitis.

Chronic constipation in childhood, giving rise to vague abdominal pain, and recurrent severe colic when purges are used, is often mistaken for recurrent appendicular trouble. Such a child put on a proper régime with suitable treatment rapidly loses his symptoms (see p. 101).

### PNEUMOCOCCAL PERITONITIS

This may be a primary infection of the peritoneum, or secondary to some other pneumococcal process in the body.

#### A PRIMARY PNEUMOCOCCAL PERITONITIS

This is a disease which as a rule affects females only, especially between the years of three and seven. It is much commoner among the poor than the well-to-do. It has been shown by McCartney and Fraser<sup>1</sup> and others that the majority of the patients have previously suffered from vulvo-vaginitis and that virulent pneumococci may be obtained from the vagina in such cases. The infection starts in the pelvis and is probably the result of an ascending infection from the vagina, through the genital tract.

**Clinical picture.**—Both before and at the onset of the infection, there is diarrhoea. Abdominal pain varies and may be misleading; usually, the pain is less than would be expected and it may be slight or absent. The onset is acute, with high fever and delirium suggesting pneumonia. Probably at this stage there is a septicæmia. Later, there is abdominal discomfort or pain with a gradually increasing tumidity of the abdomen which is tender. The temperature continues high for some days and tends to settle gradually. The full and tense abdomen gradually becomes more doughy, and localized areas of pus may be made out. The intestines are matted together and may show ladder patterns.

**Differential diagnosis.**—The acute onset may suggest appendicitis, and it is with this in mind that operation is generally undertaken. Again, if the condition has gone on for a few days the picture may somewhat resemble typhoid fever, with delirium, a tumid slightly tender abdomen and diarrhoea. The sudden onset, the delirium and the presence of pus in the peritoneal cavity and the fact that the patients as a rule are girls, should point to the correct diagnosis.

**Treatment.**—It is most debatable whether operation should be undertaken or not. McCartney and Fraser advocate immediate operation. Bellingham Smith<sup>2</sup> and others believe that operation should be left until the septicæmia stage is over and a residual abscess has formed in the peritoneal cavity. This is probably the safer course. Blood transfusions are in the author's opinion a most useful form of treatment, with laparotomy in the localized stage.

With the recent advent of the Sulpha drugs the whole question of pneumococcal infections must be revised. The author has no personal experience of chemotherapy in a condition as rare as pneumococcal peritonitis, but there is no doubt that it is indicated. (For its use see p. 183.)

**Prognosis.**—Those cases in which a residual abscess forms recover completely when this is drained. Cases with a high fever and what appears to be septicæmia stand operation badly and very few seem to recover. The mortality before the days of the Sulpha drugs was about 50 per cent.

#### B SECONDARY PNEUMOCOCCAL PERITONITIS

This may follow on a pneumonia and is included among the possible complications of pneumonia. It may be accompanied by pneumococcal infections of other serous cavities, such as the middle ear and the pericardium. When there is a general pneumococcal infection the prognosis is extremely grave. If, however, after a primary pneumonia, peritonitis appears somewhat later and the pneumonia clears up, the

<sup>1</sup> J. E. McCartney and John Fraser, "Primary pneumococcal Peritonitis," *Brit Jour Surg*, No. 36, 1922, ix, 473.

<sup>2</sup> "Discussion on Pneumococcal Peritonitis," *Proc. Roy Soc Med*, No. 7, May, 1925, xviii, 46.



Plate 3 — Radiographs of normal infants immediately after a bottle feed. Note the wind in the stomach of each infant and its presence in the large intestine from previous feeds.





Fig 1—Barium enema in a case of ulcerative colitis

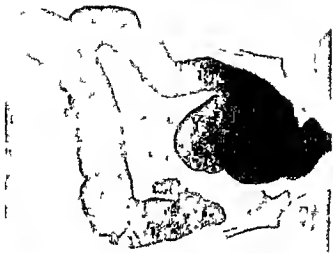


Fig 2—Normal appearance of barium enema. Note transverse colon in each case

**Clinical picture**—As a rule, inguinal hernia is present at birth or shortly after birth and is first noticed as a swelling in the scrotum much more marked when the infant is crying. When the child is quiescent the lump seems to disappear. It may be present on one or both sides. On examination, slight pressure will often replace the hernia with a gurgle, showing that it contains bowel. The author has repeatedly seen an ovary prolapsed into the sac, or the cecum and appendix prolapsed into a large hernia. The size of the ring through which the hernia has passed can be ascertained by passing the little finger up the inguinal canal. Occasionally, a hydrocele accompanies or is mistaken for a hernia and thus complicates the diagnosis.

**Irreducible and strangulated hernia.**—Very occasionally, the rupture is not replaceable. In these cases immediate operation is required. Should the rupture become firm and tense, and threaten to become irreducible and strangulate, the following procedure should be attempted. The infant is laid on a pillow with the head considerably lower than the buttocks. A feed is given to quieten it. When quite quiet and sucking, gentle pressure is exerted over the mass and an effort made to replace the rupture. An ice bag may be applied with care. Should this method not succeed, an anæsthetic is given and another attempt made. Force must not be used or the gut may rupture. In a very small proportion of cases operative treatment is necessary.

**Prognosis**—One fourth of all inguinal hernia disappear spontaneously, during the first three years of life. The remainder will require operation sooner or later.

**Treatment**—Many small illnesses are attributed to a hernia in an infant, but this is probably quite incorrect. Infants who are not treated with a truss or other appliance seem to thrive just as well as those who are treated. Nevertheless in many cases some form of retentive apparatus is indicated.

1 A woollen truss. The application of a woollen truss is not easy and the author has discarded it.

2 A rubber horseshoe truss. This is by far the easiest to apply and the most efficient. Being made of rubber it is easily cleaned and re-applied.

**Surgical treatment**—In three out of every four inguinal hernia operative treatment should ultimately be resorted to. It is best that this operation should not take place before the age when the child is clean in its habits, that is, before 18 months to two years. At this age the child is running about and there is a greater tendency due to activity, for the rupture to come down. Recurrences and mortality after operation are negligible.

### UMBILICAL HERNIA

This is much less common than inguinal hernia and is found equally in males and females. By the age of 10 or 12 years half the cases of umbilical hernia have disappeared spontaneously, while the remainder are much reduced in size. The application of adhesive strapping over a covered in halfpenny, or a rubber truss, may be recommended as treatment. In cretinism (see p. 368), accompanying the protruding tongue and general backwardness, there is a large umbilical hernia. This answers rapidly to treatment with thyroid gland. Hypotonic, flabby, weakly children, such as mongols, tend to have widely separated recti muscles and umbilical hernia. Children with rickets and coeliac disease are both liable to umbilical hernia. The treatment of the general condition of such children is essential if a spontaneous cure of the hernia is to be expected. If the rupture is of any size, surgical treatment should be undertaken as soon as the child is clean in its habits.

### COMBINED INGUINAL AND UMBILICAL HERNIA

In about one third of such cases the hernia disappear spontaneously. The other two thirds require surgical intervention.

### INTUSSUSCEPTION<sup>1</sup>

**Ætiology.**—Intussusception is slightly more frequent in boys than in girls, and is commonest between the ages of 6 and 18 months although it may occur at any age. The patients are usually vigorous, strong children. At a children's hospital it was noticed that intussusception

<sup>1</sup> S. Monrad, "Acute Invasion of the Intestine in Small Children," *Arch. Dis. Child.*, 1926, 1, 593.

generally appeared at week ends and during the holiday season, and food indiscretions have long been considered the primary cause, acting, it is thought, by causing a gastro enteritis which inflames the lymphoid tissue especially about the cæcum. The nodule of lymphoid tissue serves as a starting point from which the intussusception moves forward. A polypus or other growth will also act in the same way.

**Varieties.**—The most common form is the ileocaecal in which the ileum is invaginated into the cæcum, and passed up the ascending colon and onward. The next most common is the ileo colic then the purely enteric, or intussusception of the small intestine only. Finally, there is the pure colic type.

**Clinical picture.**—A fat, well nourished child usually breast fed, between the ages of 6 and 9 months, suddenly screams and draws up its legs, looking grey and ill. The attack may pass off to be followed quickly by another and yet another at varying intervals of a few minutes. Very often this is followed by a stool containing blood and some mucus, which may be repeated. No further fæces are passed. Between attacks of pain the child may appear quite bright although not himself. Careful palpation of the abdomen reveals a sausage shaped mass usually lying transversely across the abdomen. The mass may be soft and difficult to feel, but with continued palpation it hardens under the hand so that it is unmistakable. On rectal examination the end of the mass may be felt if it has travelled round to the sigmoid, but usually nothing can be discerned. On withdrawal, however, the finger is covered with blood and mucus.

**Prognosis.**—If the case is taken early the outlook is excellent, but the mortality rises steeply after 12 hours. After 24 hours the outlook is bad.

In a ten year period at the Royal Edinburgh Hospital for Sick Children Herzfeld<sup>1</sup> reported 15 deaths out of 107 cases, of the 15 only three occurred during the second five year period.

**Differential diagnosis.**—The commonest mistake is to confuse an acute entero colitis with intussusception. In the former, blood and mucus are present in the motions, the child is collapsed and there may be vomiting. The attacks of pain are not so severe nor so frequent. The temperature is high. An examination reveals no mass and the motions are frequent, although small. Other conditions to be excluded are Henoch's purpura and abdominal tuberculosis.

**Treatment.**—The treatment is surgical and should be instituted at once.

**Chronic intussusception.**—This usually occurs in older children but may be found in infants. A child may have attacks of abdominal pain at hourly intervals for some weeks, and on examination a mass is found. Meanwhile, motions are passed and blood is unlikely to be present. Such cases tend to recur after operation and reduction.

#### PROLAPSE OF THE RECTUM

**Incidence.**—Prolapse of the rectum is quite common, the incidence being about equal in the two sexes. It may occur at any age, but is most frequent between one and three years. The author has seen a case aged six weeks.

<sup>1</sup> G. Herzfeld, *Edin. Med. Jour.*, December 1933, xii 750.

**Ætiology and pathology.**—Contrary to expectation, diarrhoea accompanies the prolapse more often than constipation, and wasting is therefore an almost invariable accompaniment. Prolapse is frequent in cases of coeliac disease or chronic intestinal indigestion. With constant frequent stools there is much loss of the intrapelvic fat, especially in the ischio-rectal region. The rectum is not supported properly, the sphincter becomes lax and there is a protrusion at the anus, at first very slight, and finally a large extrusion, as big as a tangerine orange, may be seen. Bleeding from this surface and ulceration are common, and there may be great difficulty in replacing the mass.

**Treatment.**—The mass may be replaced by covering it with a piece of clean lint or muslin placing the whole hand over it and gently pressing. The child's attention should be distracted if possible by talking to him or interesting him in food or some other object. If necessary, an anæsthetic may be given.

Once the mass is replaced the buttocks should be strapped tightly together, the child lying fully extended. The ankles and knees should also be bandaged, and it is most useful if a long latten splint can be bandaged to the side of the child, to prevent him sitting up. The bowels should be kept well open and the motion passed in this recumbent position, thus preventing straining and recurrence of the prolapse. This treatment should be continued for one or two weeks, allowing the sphincter and to recover its tone. Treatment should be given at once for the underlying wasting. In a case of coeliac disease dieting on the proper lines should be instituted (see p. 86), and if the patient is merely a badly fed, poorly nourished child, a proper diet and tonics should be administered.

**Alcohol injection**—Probably one of the most efficient methods of dealing with rectal prolapse is by alcohol injection. This is extremely simple and cannot be termed a surgical operation. It is done by the physician. The bowel is carefully washed out and the child is given a general anæsthetic. The skin of the buttocks and anus is cleansed with ether soap and spirit. A fine needle, some 3 in. long, is used with a small Record syringe containing 1 c.c. of absolute alcohol. The finger is inserted into the anus and the point of the needle is inserted just to the outside of the point where the anal mucous membrane and the skin meet, that is, in the outer portion of the sphincter ani. The needle then passes upward and backward towards the hollow of the sacrum, the point of the finger in the rectum guiding it so that it lies well outside the rectal mucous membrane. When it strikes the sacrum it should be pulled back a little and most of the 1 c.c. of absolute alcohol injected, the remaining small portion being injected as the needle is withdrawn. The needle is then inserted on the opposite side of the anus and the same procedure followed. In this way the rectum is tacked up to the hollow of the sacrum by a small portion of scar tissue which forms rapidly. A dressing is applied over the anus, the buttocks are strapped tightly together with adhesive and the child is kept lying flat, passing his motions when recumbent. In about a week he may be allowed to run about, but he should pass his motions when lying flat for a further week or ten days. As a rule, cure is effected.

## HIRSCHSPRUNG'S DISEASE

### (CONGENITAL MEGACOLON)

This disease was first described in 1896 by Hirschsprung. The essential characteristics are progressive constipation, with great hypertrophy of the colon and rectum. True Hirschsprung's disease is congenital and begins at or soon after birth, but may be delayed until the later years of childhood. L. E. Barrington Ward<sup>1</sup> has analysed 51 cases at the Hospital for Sick Children, Great Ormond Street. Of these, 41 were boys, and 10 girls. In 19 out of 24 cases the rectum was affected as well as the colon. The parts were dilated and the walls greatly hypertrophied.

**Ætiology.**—It is thought to be caused by an over action of the sympathetic, causing a spasm of the bowel, or an underaction of the parasympathetic, thus allowing the sympathetic to over act.

**Clinical picture.**—There is progressive constipation, with infrequent motions, punctuated at intervals by bouts of diarrhoea, which are due to "overflow." Loose motions pass round the sides of the scybalous masses filling the rectum. Later, the

<sup>1</sup> L. Findlay, "Injection of Alcohol in the Treatment of Prolapse of the Rectum," *Lancet*, 1922, i, 76.

<sup>2</sup> L. E. Barrington-Ward, "The Abdominal Surgery of Children" (Oxford Medical Publications), 1937.

abdomen becomes much distended. Anæmia results. The child tends to remain small and presents a poisoned or earthy appearance although he may be well covered. **Prognosis**—There is a great tendency to complete obstruction later and vomiting takes place. A partial volvulus may form and a laparotomy may have to be performed to save life.

**Treatment**—Abdominal sympathectomy appears to be most successful. Spinal anæsthesia is advocated as a cure in some cases.<sup>1</sup>

As medical treatment the liquid paraffin preparations particularly those combined with phenolphthalein are most satisfactory. Occasionally such children require an additional dose of castor oil or senna. Infrequent washing out of the rectum with olive oil is helpful if not too long continued. Potassium bromide 5 to 15 grains thrice daily and vitamin B complex and liver given parenterally are of value (for dosage see p. 86).

## FUNCTIONAL DISORDERS

### RUMINATION<sup>2</sup>

#### (MERYCISM)

This is the term used to describe a habit developed by some infants of vomiting or spitting up their food and re-swallowing a portion of it.

**Ætiology**—Very often the food is a pleasant one such as one of the sweetened condensed milks and has been given in too dilute a form so that the total feed is greater than the stomach can contain. The result is that natural vomiting occurs at first and the infant soon acquires the trick of being able to regurgitate its food at will. It has been said that some acute digestive disturbance may initiate the vomiting and that it continues afterwards as a habit but this has not been the author's experience. It is claimed also that rumination is a family disease which can be traced through the generations. This also cannot be borne out by the author.

**Clinical picture**—As a rule the child is very much under weight thus at say six months it may weigh only 7 or 8 lb. which is very little more than birth weight. The story is that from a very early age a few weeks only the infant brought its food up in small amounts almost continuously from one feed to another. The pillow was always wet with its vomit. In many cases the regurgitation can hardly be called vomiting but rather spitting. On close inspection the infant can be seen to hollow the tongue like a funnel champing the jaws and struggling as if uncomfortable. Occasionally the back is arched and struggling as if unwell up in its mouth a small portion spilling from the corner and the remainder being slowly re-swallowed. Thus the process is repeated over and over again until a considerable proportion of the food is wasted.

**Diagnosis**—It is seldom that the clinical picture which has been described is seen by the physician or even the mother as the infant usually a girl bright and clever will not ruminate while anyone is present. The process is best studied by placing a screen round the head of the bed and peering over the top. The infant when she thinks herself alone will go through the process.

**Treatment**—There can be little doubt that rumination is a purely

<sup>1</sup> M. Hawksley, Hirschsprung's Disease, *Proc. Roy. Soc. Med.*, Sept. 1913, **xxvi**, 536.

<sup>2</sup> Donald Paterson, "Rumination (Merycism) in Infants," *Practitioner*, Dec., 1902, **ccxi**, 330.

*functional complaint* The *spitting and vomiting* will cease in the following circumstances —

1 If between feeds the child is watched and interested, held up to look out of the window, played with and talked to

2 If, by some mechanical means, the jaws are prevented from opening and champing A small skull-cap with broad strings passing from the ears under the chin and fastened on the top of the head again, thus firmly clenching the jaws, will act as a deterrent Unless the mouth can be easily opened and closed the infant fails to get the pleasure from ruminating which makes it worth while

3 If the food is thickened with some starchy preparation, so that it is small in bulk and firm in consistency such starchy substances may be Benger's or Savory and Moore's Food, Robinson's Patent Groats, Cream of Wheat, Semolina, Farina or Wheatena The author has found Benger's Food of great service in such cases Cod or halibut liver oil is particularly important for such infants, who usually make such rapid progress that they tend to develop acute rickets They may, in fact, require a larger dose than the normal infant

4 If during the process of rumination a drop of quinine solution is placed in the open mouth The face of the child shows its disgust, and for the time being all efforts at rumination cease

**Prognosis**—On thickened feeds children given to rumination gain weight rapidly The mortality is extremely low

### AEROPHAGIA

#### (WIND SWALLOWING)

Swallowing wind is normal and physiological in every infant, breast or bottle-fed X rays show a normal bubble of air at the fundus of the stomach (Plate 8) Brennemann has shown that the thicker the feed the less the wind swallowed with it

Every infant should therefore be held up for from 15 to 20 minutes after each feed, until the wind is broken or eructated twice if undisturbed sleep is to follow Unless the child is properly held up, the air is either eructated lying down, in which case part of the feed is brought up at the same time and wasted, or it passes through the pylorus into the bowel, causing colicky pains, distension and increased peristalsis

Although slight degrees of wind swallowing are physiological, the habit may very easily become pathological An extreme example of this is seen in infants who are grossly underfed on the breast or bottle, or in infants who lack the inborn ability to suck properly, mouthing the teat rather than fastening on to it with tight suction

One of the commonest mistakes made is to give the feed through a teat having too small a hole Mothers and nurses are taught that wind is swallowed because of too fast a teat, but this is a fallacy At each suction a small quantity of air is swallowed, and if, say, 500 suctiones are required for the infant to take the feed, more wind must necessarily be swallowed than for, say, 300 suctiones with a larger hole With a very fast teat, gulping and spluttering result, but the hole should be of a sufficient size to allow the infant to get the whole feed in from 7 to 10 minutes Then

from 10 to 15 or even 20 minutes should be spent in holding the infant up

**Treatment**—1 Offer the infant a sufficient supply of food for his needs. If breast fed make sure that he is not being underfed.

2 See that he gets his food easily. Enlarge the hole in the teat and do not leave him at the breast longer than is necessary for it to be thoroughly emptied. This can be judged by doing test feeds.

3 Hold the infant up for sufficient time after each feed for him to break his wind twice.

4 For a week or two after commencing treatment give the infant (three months of age) a grain of chloral three or four times daily so that he approaches the feed in a less frantic condition.

## HEPATIC DISEASE

### JAUNDICE<sup>1</sup>

McVee classifies jaundice under the following headings —

1 *Obstructive hepatic jaundice*—This is unusual in childhood as stone in the common bile-duct growth in the pancreas or glands pressing on the common bile-duct are comparatively rare. In such cases the van den Bergh test shows a strongly positive direct reaction. The obstruction is more commonly congenital and may or may not be accompanied by congenital obliteration of the bile ducts.

2 *Toxic and infective hepatic jaundice*—The best example of this is epidemic infective hepatitis. Spirochaetal jaundice (Weil's disease) is another. Phosphorus and chloroform poisoning also come into this group.

3 *Hæmolytic jaundice*—In this group the van den Bergh reaction is usually indirect and delayed. There is some bile in the stools. The best examples are simple physiological icterus neonatorum, icterus gravis neonatorum and acholuric family jaundice.

In infants the commonest types of jaundice are —

- (a) Simple icterus neonatorum
- (b) Icterus gravis neonatorum
- (c) Icterus due to a liver infection secondary to neo natal sepsis such as a septic umbilicus
- (d) Congenital obliteration of the bile ducts
- (e) Congenital syphilis (rare)

In older children, the commonest types are —

- (a) Epidemic infective hepatitis (catarrhal jaundice)
- (b) Obstructive jaundice due to cirrhosis and glands
- (c) Acholuric family jaundice

### EPIDEMIC JAUNDICE

#### Acute infective hepatitis (Catarrhal Jaundice)

Although sporadic cases may occur the disease is essentially seen in epidemic form in schools and other institutions.<sup>2</sup>

<sup>1</sup> Robert Hutchison and Donald Paterson "Infective Hepatitis with Chrysoids" *Brit Jour Child Dis.*, 1921, xxi, 275.

<sup>2</sup> A. A. Lilney "Epidemic Catarrhal Jaundice in School Children" *Brit. Med. Jour.*, April 2, 1937 I, 703.  
H. Barber "Infective Hepatic Jaundice," *Brit Med Jour.*, Jan. 9 1937 I, 67.

During the war its incidence has greatly increased. It is thought to be due to a virus infection.

**Incubation period.**—This appears to be about a month although in some cases it is shorter, or may be up to two months.

**Age incidence.**—Children above the age of six years and young adults are more commonly affected although occasional cases are seen between the first and second year of life.

**Clinical picture.**—The onset is sudden with headache, fever and malaise. Vomiting and quite severe abdominal pain are also frequent. At this stage there is no definite clue to the disease and it is often termed gastric influenza. In three or four days or longer, depending on the severity of the symptoms, jaundice appears first in the sclerotics and then becomes widespread and bile is obvious in the urine. As a rule, the diagnosis is easily made by the seventh day. Both the liver and spleen are easily palpable and the stool becomes light and finally almost white. The Van den Bergh test gives a strong, positive direct reaction on the blood serum. The plasma phospha-tase which is normally 5 to 15 units is as high as 40 units and falls to normal by the second or third week.

Dislike of food with nausea and vomiting may continue for two or three weeks. The tongue is coated and the patient feels low and dispirited. As a rule the temperature has subsided in one to two weeks. Convalescence is slow and it takes in all about 6 weeks to make a clinical recovery. The sedimentation rate is helpful in assessing the recovery as it remains raised during the acute and sub-acute phases.

**Pathology.**—Roholin and Iversen<sup>1</sup> described the pathology of the liver as a diffuse hepatitis characterized by inflammatory phenomena with the predominance of mononuclear cells, destruction of the trabecular arrangement of the liver cells, necrotic disintegration of the parenchyma cells and a varying proliferation of connective tissue. The interlobular bile ducts were normal. The work of McMichael and Dible (1948) confirms this pathology.

**Treatment.**—The patient should be put to bed and kept there until the appetite has returned, the stools and urine are normal in colour and all traces of jaundice have disappeared. The bowels should be kept open with some mild vegetable or saline aperient.

**Diet.**—This should be low in fat and high in milk protein (casein) and carbohydrate. Glucose fruit drinks should be offered, and eggs and butter excluded during the acute and sub-acute stages. Until the liver has recovered carbohydrates alone can be efficiently utilized by the body. If fats are prematurely re-introduced into the diet, progress is likely to be retarded. Cereals for breakfast with fresh or stewed fruit, vegetables and gravy, followed by stewed fruit for dinner and some cereal or crisp toast with weak tea at tea time will be found suitable for these cases. As the appetite returns, the patient improves, and fish or chicken may be gradually added to the diet. Glucose sweets and powdered glucose in fruit drinks should be given between meals, in order to raise the caloric

<sup>1</sup> K. Roholin and P. Iversen, *Changes in the Liver in Acute Epidemic Hepatitis*, Acta Path. Microbiol. Scand., 1959, A71, 427.



intake, and particularly to supply the liver cells with the most easily digested and utilized form of carbohydrate. Skimmed dried milk (M O F house hold dried milk) is particularly suitable for these cases.

**Convalescence**—From the nature of the liver lesion it is obvious that many weeks may pass before the organ has completely recovered. A period of six to ten weeks should therefore elapse before a child is sent back to resume strenuous school life.

#### ACUTE OR SUBACUTE YELLOW ATROPHY

This is an extremely rare disease which starts like an attack of ordinary epidemic jaundice the symptoms however being much more severe with headache delirium vomiting diarrhoea and high fever.

**Etiology**—The disease is commoner in older children and young adults. The cause is unknown. Some cases follow anaesthetics and phosphorus poisoning others occasionally what appears to be an acute hepatitis.

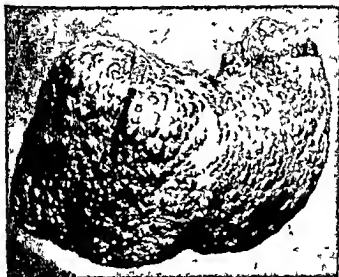


Fig 17—Hepatic cirrhosis in a child aged 6½

**Pathology**—The liver cells show acute necrosis with an attempt if the case lasts any length of time at regeneration budding out being most noticeable in the region of the bile canaliculi.

**Clinical picture**—The child who has been drowsy and vomiting becomes gradually delirious and jaundiced the urine being deeply bile stained and the motions pale with diarrhoea. What was at first thought to be catarrhal jaundice is now seen to be a grave illness. Delirium is present. The liver which at first was quite palpable shrinks rapidly beneath the costal margin and the spleen cannot be felt. There is a tendency to hæmorrhages in the mucous membranes and purpuric patches on the skin. Air hunger and coma rapidly supervene death taking place in the severe cases.

#### CIRRHOSIS OF THE LIVER

It is well for the student to realize the relative frequencies of the different types of cirrhosis. These can be shown by an analysis of 46 cases from the Hospital for Sick Children, Great Ormond Street London during the years 1910-1925<sup>1</sup>

<sup>1</sup> F J Poynton and W G Wylie. Hepatic Cirrhosis in Children, *Arch. Dis. Child.* No. 1, 1926: 11

	Cases	
Congenital biliary cirrhosis <sup>1</sup>	92	
Obliteration of the larger bile ducts	15	Survived 6 months
Ducts patent	7	2 survived for one year
Syphilis	19	All died under 8 months
Portal cirrhosis	3	
(2 aged 9 years and 1 aged 3 years)		
Acute yell w atrophy	1	
Banti's disease	1	

(A) Biliary cirrhosis<sup>1</sup>—(1) Congenital cirrhosis with or without congenital

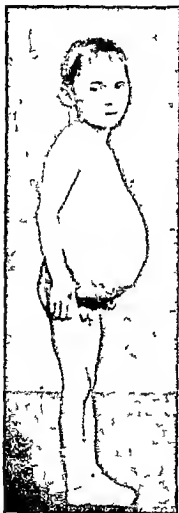


Fig 16 Case of cirrhosis of the liver with ascites

obliteration of the bile ducts.—It is impossible to tell on clinical examination whether the extra hepatic bile-ducts are obliterated or not.

This is a rare condition. Shortly after birth the child is seen to be slightly jaundiced and at first may be considered to be suffering from icterus neonatorum only. Then it is noticed that the jaundice is becoming more and more marked, the stools continue light in color and the urine is deeply stained with bile. The sclerotics have an icteric tinge. The spleen is as a rule palpable and the liver which is down several fingers breadth below the costal margin is hard and resistant on palpation. The blood-count shows no erythroblastosis. The van den Bergh test shows an immediate direct reaction characteristic of obstructive jaundice.

In 15 cases seen at Great Ormond Street jaundice and pale stools commenced in six cases at birth, three within the first week, five at the third week, and one at the seventh week. Several cases were seen which, throughout life, had a slight trace of bile in the stools.

The feature of the pathology is a uni- and intra-biliary cirrhosis. Even when there is no extra-hepatic obstruction of the bile ducts, the liver still shows some type of diffuse intra-biliary cirrhosis.

Ladd<sup>2</sup> of Boston investigated 45 cases of congenital obliteration of the bile ducts. He considered that in 15 of these cases operation was warranted and 9 recovered after a plastic operation on the common bile duct joining it up to the duodenum or joining up the gall bladder to the duodenum. In 8 cases of the 15 there were however none showed any signs of operative possibilities. Nevertheless, in view of Ladd's series, it appears that an exploratory operation is justified in all cases of this condition.

(2) Non obstructive hypertrophic biliary cirrhosis (Hanot syndrome).—This form is unusual in childhood. No cases occurred in the Great Ormond Street series. It is a chronic infection characterized by permanent jaundice, with febrile disturbances and exacerbations of the icterus, recurrent abdominal pain, considerable enlargement of the liver and spleen

absence of ascites, but dilatation of the superficial abdominal veins.

<sup>1</sup> P. H. Evans, "Biliary Cirrhosis," *Arch. Dis. Child.*, June, 1930, xiv, 89.

<sup>2</sup> "Congenital Obstruction of the Bile Duct," *Ann. Surg.*, 1935, li, 712.

The liver is dark green, and shows a finely granular surface. The cirrhosis is of the extra- and intra-lobular type, with a great increase in the number of small intrahepatic bile-ducts.

(3) **Acquired obstructive biliary cirrhosis**—This is extremely rare in childhood. Causes such as a stone in the bile duct, new growth in the head of the pancreas and tuberculous glands at the hilum of the liver are given. No case was seen in the Great Ormond Street series.

(B) **Multi-lobular (portal) cirrhosis**—This is a rare disease in childhood, and most frequent after the age of 8 years. (There were three cases in the Great Ormond Street series.) The aetiology is unknown but it is supposed to be due to some irritant or toxin absorbed from the intestinal tract, and both alcohol and vinegar have been known to produce it.

*Symptoms*—The onset is insidious. The abdomen is enlarged, although the chest, arms and legs become thinner. The child tends to be dwarfed, probably due to some defect in the vitamin A storage of the liver. The liver is enlarged and hard and there may be ascites. Telangiectases appear over the face and body, and the veins are enlarged over the abdomen. Oedema of the extremities appears. Jaundice may be present, but is not a feature.

*Pathology*—A multilobular cirrhosis is found and there is hypertrophy or proliferation of the liver cells in an attempt to make good damaged tissue. This gives a nodular appearance macroscopically (hobnailed liver).

*Clinical course*—After a varying and indefinite period, sometimes years, the child develops mild headache, oedema, delirium, twitching and coma with death.

(C) **Syphilitic pericellular cirrhosis**—This was the second commonest in the series of cases, with 19 examples. It is said that from 35 to 65 per cent. of all syphilitic infants born alive show this type of liver cirrhosis. The infection is conveyed from the mother via the umbilical veins direct to the infant's liver. The majority of stillborn infants show this complaint.

*Pathology*—The appearance in the early stages is one of diffuse round cell infiltration, resembling milky gummatum. Later, these give rise to diffuse pericellular fibrosis.

*Clinical picture*—The liver and spleen are enlarged. Jaundice and pale stools are not marked. The infant wastes, ascites appears, and secondary infections cause death within 9 months as a rule.

## CYCLICAL VOMITING<sup>1</sup>

### (ACUTE AND CHRONIC ACIDOSIS OR KETOSIS)

Acidosis or ketosis is characterized by the presence of acetone bodies in the urine, bloodstream and breath.

**Ætiology.**—Acidosis or ketosis must be considered merely as a symptom with a number of causal factors, rather than as a disease in itself.

1 *The result of infections*—All febrile illnesses or infections are accompanied by the production of acetone bodies. In ordinary practice the commonest of these is a naso-pharyngeal infection.

2 *The result of a badly balanced diet*—In our present state of knowledge we must look upon carbohydrates as the fire which burns up the fat, and any deficiency in carbohydrate or excess of fat will leave a certain amount of fat unburnt, with the result that its breakdown products, the acetone bodies, are formed. The commonest fats in excess in the diet of a child are milk (cream) and eggs.

3 *The result of over exertion and over excitement* (physical and nervous exhaustion)—Physical and nervous energy is probably derived from the glycogen stored in the muscles and liver. After an excessive amount of exercise or mental strain, excitement or shock, these stores of glycogen are

<sup>1</sup> Donald Paterson, "So-called Acidosis Attacks," *Lancet*, April 20, 1935, i, 317.

exhausted and the body fat is drawn upon, with the result that ketone bodies are produced and the child is in a state of acidosis or ketosis

4 In some children there is an *inborn tendency* towards acidosis which is undoubtedly hereditary. This hepatic insufficiency can be handed from father to son, and in most of such families one or other parent it will be found suffered from cyclical vomiting or gastric attacks as a child

5 *Migraine* should not be overlooked as a cause

**Pathology.**—Nothing is accurately known of the pathology of this condition beyond what has been already stated. If the acidosis is untreated and continues long enough death may result, and the liver will be found to have undergone an extreme degree of fatty change

**Symptoms and diagnosis**—Cyclical vomiting must be considered an extreme manifestation of the acidosis state. The vast majority of cases are much less well marked. Typical examples of cyclical vomiting are, as a rule boys than hypotonic children who stand badly, as if tired, with shoulders drooping and the pelvis pushed forward. They are over bright children, often only children

The effects of cardio vascular instability are seen in a tendency to flush easily, or to have pale turns and collapse or faint. The metabolic processes in these children are easily upset so that they show gastric or liverish attacks with infections, dietetic errors or over exertion or excitement. The vomiting may begin acutely even sips of water being returned, the breath is sighing and there is air hunger. The child becomes utterly prostrate, and presents a most alarming picture but with proper treatment recovers in a most dramatic fashion from what appears to be a severe illness. He tends to run temperatures with slight provocation, and the tongue is chronically dirty unless a proper anti ketogenic (anti acidosis) diet is adopted

**Treatment.**—During an acute attack the child should be put to bed. A careful clinical examination should be made to ascertain the cause of the vomiting the temperature being taken and particularly the throat examined. If there is a tonsillitis or otitis media, one of the Sulpha drugs should be given, and any other underlying cause, such as for example migraine (see p 125) or pyæmia (see p 200), should be appropriately treated

**Dehydration**—The vomiting may be severe, and rectal saline, with 5 per cent glucose should be given particularly if the child is constipated. The dehydration and collapse may be so severe, however, that an intra venous drip of saline and glucose, or Hartmann's solution, with glucose if necessary should be given in which case all fluid by the mouth should be withheld, to prevent alkalosis

It is a mistake, particularly in younger children to take such cases lightly. If the vomiting is allowed to continue, collapse and death may follow, or there may be some unsuspected gross organic disease

Sips of glucose fruit juice drinks should be given as soon as the child is retaining its fluids. Glucose sweets should be offered. Sometimes carbohydrates in the form of dry biscuits or crisp toast, with honey or jam, are retained best. It is a mistake to give bicarbonate of soda after the attack has properly started

*Between attacks*—Sources of infection, such as bad teeth, septic tonsils and infected ears, should be dealt with.

The diet should be perfectly normal, including a pint of milk daily. It is a mistake to keep such children on a low fat diet, particularly a milkless one, as they tend to contract more frequent infections and the teeth are often permanently damaged.

*Rest*—These children require much rest and a short day. To give breakfast, tea and supper in bed is often the best way of ensuring the extra rest. Kindergarten school is often a great success in conserving energy.

*The bowels*—It is particularly necessary that such children should not be allowed to become constipated, and milk of magnesia, Eno's fruit salts, or Salixtae are particularly suitable if given an hour before breakfast, on a completely empty stomach.

### MIGRAINE<sup>1</sup>

Migraine in young children often passes undiagnosed.

**Ætiology and pathology**,—There are various theories of its causation, and certainly it is highly familial, passing from one generation to another and alternating with asthma, hay fever and eczema in some individuals. The following are said to be factors in its production.

- 1 Allergy—sensitivity to foods or inhalants
- 2 Infections—particularly of the naso pharynx or urinary tract
- 3 Eye strain.
- 4 Nervous exhaustion

**Symptoms**,—The term 'abdominal migraine' is sometimes used to explain abdominal pain accompanied by vomiting and headache which recurs every few weeks. Fever is usually present. As the child approaches puberty the headache is accentuated. He complains of pins and needles in the fingers and tongue, and the vision is affected. A hemi-cranial headache is common, and dislike of light and violent vomiting follow. The whole thing may last a few minutes or some hours. A careful examination may reveal a sore throat or inflamed ear.

**Differential diagnosis and treatment**—The greatest care must be exercised to ensure some serious organic disease does not occur under the guise of migraine. Nephritis, high blood pressure, brain tumour with papilloedema, recurrent appendicitis or some abnormality of the genito-urinary tract must all be carefully excluded. Once the diagnosis has been arrived at, infected teeth and tonsils should be dealt with and an appropriate dose of ergotamine tartrate (Femergin) should be given. A suitable dose for a child aged 6 years is one tablet of Femergin (0.001 gm.) to be given at the commencement of an attack and repeated in half an hour if there is no relief.

Skin testing for food sensitivity and the exclusion of certain foods has not been successful in the author's hands.

<sup>1</sup> 'Relief of Migraine', *Edin. Lancet*, Feb. 13th 1943, 1, 209.

## CHAPTER VIII

### DISEASE OF THE RESPIRATORY SYSTEM

#### CONGENITAL MALFORMATIONS

##### CONGENITAL CYSTIC DISEASE OF THE LUNG<sup>1</sup>

CONGENITAL cystic disease of the lung is a very rare condition. It may take the form of a single large cyst, filling almost the whole of one side of the chest. There may be very little embarrassment, and single or multiple cysts may be discovered quite by chance. In such a case the prognosis appears reasonably good.

The other type is a multiple cystic disease, of one or more lobes either uni- or bilateral. In this condition the symptoms, which are cough and dyspnoea date from shortly after birth. As the child gets older, the condition resembles bronchiectasis. There may be sputum and the diagnosis can be confirmed by X rays before and after lipiodol. The prognosis appears good as many such cases are seen later in life attending adult chest hospitals as out patients (Young) and living a moderately normal existence.

##### CONGENITAL LARYNGEAL STRIDOR

This is more commonly found in girls than in boys, and it is present from birth.

**Clinical picture and diagnosis**—Very shortly after birth a noise on inspiration is noticed. It is a crowing noise accompanied by an indrawing of the intercostal spaces and of the episternal notch, worse when the child is roused than when quiescent. The physical condition is as a rule excellent and the mentality normal. The voice and cry are clear. The condition must be differentiated from papilloma, where the voice is hoarse and the symptoms come on some time after birth and from laryngismus stridulus which accompanies acute rickets.

**Ætiology and pathology**.—Occasionally, the stridor may be associated with under development (hypoplasia of the mandible (micrognathia). In such cases, the tongue appears to fall back and give rise to the inspiratory stridor. Laryngoscopic examination may show a narrowing and flabbiness of the aryteno epiglottic folds, which tend to collapse and obstruct inspiration.

**Prognosis and treatment**—As a rule, the symptoms pass off about the age of one year, and certainly by two years, and there is no further sign of the condition. No active treatment appears to be of any benefit nor does it seem necessary.

##### CONGENITAL ATELECTASIS OF THE LUNG

The normal condition of the lung before birth is one of collapse. At birth with inspiration, the lung becomes inflated and the process of expansion takes place within a day or two especially with vigorous crying. Failure of the lung to expand (atelectasis) is uncommon.

<sup>1</sup> C. E. Field, "Cystic Conditions in the Lung, *Proc Roy Soc Med*, Sept., 1913 xxvii, 534

**Etiology and pathology.**—The condition is most commonly due to some congenital malformation of the bronchi, a lobe or portion of a lobe remaining cut off from the bronchial tree. Such an area may present a cystic appearance *post mortem*, and may be free from air. Another cause is blocking of the bronchus leading to that portion of the lung by inspissated mucus or some foreign body. If the infant is feeble or premature it may have insufficient strength fully to aerate the whole of its lung tissue.

**Clinical picture.**—An infant with congenital atelectasis may present few or no symptoms, but if it be premature or weakly its other symptoms may be aggravated by the pulmonary condition. Sudden 'blue turns' or attacks of dyspnoea, such as are seen in congenital heart disease, may occur. An infant with extensive atelectasis, but otherwise perfectly normal, sooner or later presents a picture of respiratory insufficiency, dyspnoea on slight exertion, a tendency to cyanosis and a cough, with bronchitis to a greater or lesser degree. On examination, there is dullness on percussion over the affected part, and displacement of the mediastinal contents towards that place. On auscultation, the breath sounds are tubular, being conducted by the solid substance of the lung. A ray examination confirms the picture. In older children it is difficult to differentiate between the appearances described and those due to pulmonary fibrosis following on unresolved pneumonia. In a new born infant, however, a pulmonary fibrosis is uncommon.

**Prognosis.**—If the atelectasis is only of a small portion of a lobe, it is not incompatible with life and may even escape detection for many years. If a lobe or more is involved however, the symptoms are sufficient not only to attract attention but to cause serious ill health. A severe atelectasis in a new born infant often leads to death.

**Treatment.**—Where there is some congenital malformation nothing can be done, but if mucus is present it may be loosened and its removal aided by the inhalation of 5 per cent  $\text{CO}_2$  in oxygen through a nasal catheter. Inversion of the child helps to drain away obstructing mucus. The mode of life of such children is like that of the child with congenital heart disease, and symptoms must be treated as they arise.

#### LARYNGEAL PAPILLOMA

This is more commonly found in girls than in boys. Whether it is present from birth is not known, but the first symptoms appear at the age of about 2 or 3 years.

**Clinical picture.**—The onset is very gradual, the voice being slowly lost. There is usually only a huskiness at first, then gradually complete loss of voice occurs. Later, symptoms of inspiratory and expiratory obstruction appear, which may become acute and threaten asphyxiation.

**Pathology.**—Growing from the vocal cords, or wall of the larynx close to the vocal cords, several small papillomata are usually found. When removed they tend to recur repeatedly, but some tend, on the other hand, to disappear spontaneously.

**Treatment.**—This is largely surgical, and operative treatment should be delayed as long as possible, as the older the child the larger the larynx and the greater the opportunities of performing a successful operation. X rays and radium treatment are claimed as successful in a minority of cases. Tracheotomy and intubation may have to be done, with operative treatment some time later.

**Prognosis.**—The prognosis is most unsatisfactory. Patients operated on early die of pneumonia, as a rule, and when operation is left until later, such extensive removals must be done that permanent loss of voice results. Where tracheotomies are performed, much trouble is given by each scar. Strenuous efforts should undoubtedly be made to treat with radium.

#### CROUP

This term is commonly applied to two different conditions —

- 1 Spasmodic croup or catarrhal laryngitis,
- 2 Diphtheritic croup

#### Spasmodic Croup

**Etiology.**—Spasmodic croup is due to a catarrhal infection, most commonly the *Micrococcus catarrhalis*, extending from the naso-pharynx to the vocal cords and larynx. There is swelling and congestion of the vocal cords, which impede their function. In children, it is commonly accompanied by a greater or lesser degree of spasm of the laryngeal muscles.

**Age-incidence.**—Spasmodic croup most commonly occurs between the ages of 6 months and 3 years.

**Clinical picture.**—During the afternoon the child may have a slight cough and be a little feverish. After being put to bed he awakes and attracts attention by his noisy breathing. There is both an inspiratory and an expiratory stridor. Cyanosis is often present to a marked degree. All the ordinary and extraordinary muscles of respiration are over acting. The general constitutional upset is not great, and immediately the attack is over the child seems himself again. Spasmodic croup appears, therefore, to consist of two separate factors: first the catarrh of the vocal cords and secondly the nervous spasm of the larynx. The latter is the more important in producing the clinical picture.

**Prognosis.**—Simple spasmodic croup is seldom fatal. Once an attack has occurred other attacks are to be expected.

**Differential diagnosis.**—It is necessary to differentiate between laryngeal diphtheria, laryngismus stridulus (a symptom of tetany or acute rickets) and croup. In laryngeal diphtheria the constitutional upset is severe and the colour grey. The onset is seldom sudden, often occurring in the morning or during the day. An examination of the throat, as a rule, shows accompanying pharyngeal diphtheria or nasal diphtheria. When there is any doubt whatever antitoxin should be administered at once. In laryngismus stridulus the stridor is inspiratory, and between attacks the breathing is quiescent. The face of the child with croup is, as a rule, flushed and shows the presence of some infection of the respiratory tract, a fact which is helpful in distinguishing it from diphtheria. Throat swabs should be taken in all doubtful cases.

**Treatment.**—Since the spasm of the larynx is much more severe during the child's waking period than during sleep, every effort to induce sleep should be made. A grain of chloral at six months of age, two grains at one year, and an additional grain for each year of the child's life, can be given safely three or four times daily. Where it is difficult to induce the child to swallow it, the chloral may be given rectally. Much relief is obtained if the child is propped up in bed with a steam kettle containing Eriar's balsam or a cresolene lamp. Care should be taken, however, that the steam is not on for more than 15 minutes to half an hour in three hours, as it is easy to overdo this treatment. The application of warm flannels or Antiphlogistine to the neck is recommended. The bowels should be kept well open and the diet should be light, as recommended for fever patients (see p. 829). Tracheotomy or intubation is indicated in cases which continue to show acute spasm for more than 24 hours, particularly if the cyanosis is marked. Children tend to tire suddenly and get acute cardiac dilatation. Croup should therefore be taken seriously in all but the mildest cases. Allergy should be borne in mind as a possible etiological factor, and laryngo-tracheal bronchitis may closely simulate croup.

#### DIPHTHERITIC CROUP

Differentiation between this form of croup and spasmodic laryngitis is sometimes extremely difficult. Where there is any doubt, antidiphtheritic serum, in doses of 4,000 to 8,000 units, should be administered at once. It may be necessary to perform



tracheotomy in severe cases. Intubation however is to be preferred in institutions as the sequelæ of tracheotomy are most undesirable

### UPPER RESPIRATORY TRACT INFECTIONS ACUTE RHINITIS (THE COMMON COLD)<sup>1</sup>

**Etiology.**—Dochez<sup>2</sup> ascribes this to a virus infection. It is highly infectious, and passes rapidly from one child to another. A "cold" appears to give very little acquired immunity to the individual. Because of this, repeated colds can and do affect susceptible subjects.

**Clinical picture.**—The incubation period may vary from a few hours to thirty-six hours. The prodromal symptoms are sneezing and a feeling of fullness in the nose and throat. There is usually some headache, and there may be a tendency to shivering. The eyes are puffy and there is a mild conjunctivitis. The nasal mucus is involved and a profuse watery discharge takes place from the swollen mucous membrane. As a rule, in infants and young children there is some fever up to 102° F. In the tiny infant there may be very little local reaction (that is not much sign of a cold) but "symptomatic diarrhoea and vomiting" may result. The infection may spread to the larynx or ears. In an average case in two or three days the secretions become thicker and less severe and gradually the inflamed nasal mucous membrane subsides. The whole process takes about a week.

**Prophylaxis.**—The affected individual should go to bed at once thus being isolated from the family. Plenty of fresh air particularly where two or more children occupy the same bedroom is recommended.

Helen Mackay has shown that anæmic infants and children have a much higher incidence of colds and infections than those who are full blooded. A diet rich in iron and liver is therefore indicated. Allergy should be borne in mind: sometimes so called "colds" are in fact attacks of rhinorrhœa, and not true virus infection. Careful skin testing and a careful history will bring out this point. In rhinorrhœa sneezing is as a rule, excessive and there is generally no temperature.<sup>3</sup>

**Treatment.**—The patient should be put to bed or in summer time, placed in its cot out of doors, both for his own sake and the sake of others. The bowels should be kept well open and plenty of drinks of fruit juice offered. The nose should be wiped repeatedly and the child taught when possible, to co-operate by blowing into a handkerchief without compressing the nostrils. A few drops of 2 per cent argyrol, or 1 per cent menthol in liquid paraffin, or blueo ledrin drops, will help to keep the nasal passages clear. A benzadrine inhaler is helpful in the older child. Infants with colds should be nursed as far as possible in the upright position, or with the head raised, and not fed lying down. There is a great tendency for the catarrh to seep by gravity into the ears. No cold should be treated lightly, particularly in the infant, as it may be the starting point of a chain of events of far reaching importance. To the weakly infant a cold may lead to refusal of the breast, with premature weaning.

<sup>1</sup> J. Crooks, in Carol Ratten and Thrusdell's, *Diseases of Children* (Arnold's), 1931 p. 397.

<sup>2</sup> Dochex, *et al.* *Lancet* 1931 B 54.

<sup>3</sup> H. M. Mackay. *Nutritional Anæmia in Infancy with Special Reference to Iron Deficiency*. Med. Res. Council Report, 1932.

One grain of powdered *aspirin* may be given to a feverish infant, three times daily, with benefit. In severe colds with fever, one of the Sulpha drugs should be given. It will be found to shorten and modify the attack and help to prevent complications such as otitis media.

### ACUTE FOLLICULAR TONSILLITIS

**Ætiology.**—This is usually due to a streptococcal infection, often of the hæmolytic variety. Occasionally a staphylococcal or pneumococcal infection may be the cause. Other rare organisms, such as the bacillus of Pfeiffer, are also found.

**Mode of infection.**—As a rule, tonsillitis is acquired by droplet infection from some other individual. Occasionally it is acquired from milk, as in the Brighton epidemic some years ago.

**Clinical picture.**—The onset is sudden, the first symptoms may be those of the common cold—lassitude, fullness in the nose and throat, headache and shivering. The throat is sore, and the child often refuses to eat or drink. It is, however, unusual for a child to complain of sore throat before the age of five or six years. Most parents fail to realize this, and are much surprised when the diagnosis of tonsillitis is made. In every case of illness in an infant or child, it is necessary, therefore, to examine the throat carefully.

**Method of inspecting the throat.** It is best to wrap the child round with a blanket or shawl confining the arms tightly. The nurse or mother should sit the child upright on her knee, and place one arm round the child's body while the other arm hand on forehead, holds the head firmly against her chest. The physician is now free to hold his torch in one hand and a teaspoon or wooden spatula in the other hand. To get a complete view it may be necessary to make the child "retch" somewhat, in order that the lateral surfaces of the tonsils shall protrude forward.

It will be noticed that the anterior pillars of the fauces are red and œdematous, and that the tonsils themselves are dark red and engorged. At intervals over the tonsil itself, protruding from the crypts, small points of pus will be seen—perhaps from four to a dozen on each side in a severe case. Very often the tonsillar glands at the angle of the jaw are enlarged, but they may not be in the first or second attack. The tongue is coated, and tends to be dry, the breath is unpleasant. There may be a short cough, or a continual clearing of the throat, particularly in nervous children. If solid food is taken there is often a tendency to retch and vomit. The temperature is high, and the face flushed.

In infants there may not be a violent reaction to the infection. Much less is to be seen locally, but the general upset is greater and more widespread.

**Course.**—In three or four days there is a decided improvement, and by the tenth day the throat condition has largely cleared. Much, however, will depend on the age of the child and the nature of the infection. With modern treatment, the course may be much shorter, particularly in first attacks and where the glands and ears have not become involved.

**Complications.**—There may be abdominal pain, almost invariably due to coincident enlargement of the mesenteric glands. Occasionally, an

inflammation of the appendix is found. The commonest complication is symptomatic vomiting and diarrhoea, particularly in infants. (See Symptomatic Diarrhoea, p 103, and Infective Acidosis Attacks, p 129) Earache, pan sinusitis and mastoid disease are also complications (see p 138). Cervical adenitis is dealt with on p 136. Peritonsillar and retropharyngeal abscess are occasional complications (p 135).

**Treatment.** *Drugs*—With the introduction of the Sulpha drugs there is no doubt that this complaint has been much shortened. The temperature subsides, in many cases a day or two after treatment starts. (For dosage see p 388) The bowels should be kept well open with a routine dose of milk of magnesia or one of the saline aperients. Powdered aspirin in appropriate doses, used as a gargle, and then swallowed greatly relieves the pain.

*Diet*—Much bland fluid, containing well sweetened fruit juice, should be offered. It is often best to keep a jug of fruit juice alongside the bed to act as an invitation to the child to drink more. Milk should not be pressed in a case of follicular tonsillitis. When the appetite returns milky drinks to which have been added one of the starchy preparations mentioned on p 403, may be offered.

*General nursing*—Tepid sponging is indicated if the temperature is very high. The mouth should be cleansed several times daily with glyco-thymoline, listerine, glycena or borax, or some other mild antiseptic. Painting the throat is not a great success in the majority of cases as it usually makes the child vomit up the last meal. If at all it should be performed when the stomach is empty. A weak solution of iodine may be used once with benefit. The child should be kept in bed until the temperature has been normal for two or three days. A tonic should be given, such as cod or halibut liver oil and malt, one teaspoonful three times daily, or some pleasant iron preparation, such as the saccharated carbonate of iron, half a teaspoonful night and morning, or Ferrodic (Allen & Hanbury), 1 teaspoonful three times daily.

#### RECURRENT NASO-PHARYNGITIS<sup>1</sup>

Infections of the nose and throat, or upper respiratory tract infections are extremely common in childhood. Nothing plays so large a part in producing the minor ailments of infancy and childhood.

**Ætiology.**—The infecting organisms may be the *Micrococcus catarrhalis*, the pneumococcus, streptococcus or influenza bacillus. These infections are easily passed from one member of a household to another and there can be little doubt that sore throats and infected adenoids with nasal discharge are commonly contracted from some other child. They may, however, be brought about by warm, stuffy rooms, when the normal organisms of the nose and throat may obtain a hold on the nasal mucous membrane. Accompanying measles and scarlet fever there is usually well marked infection of the nose and throat.

**Pathology**—The inflamed lymphoid tissues of both adenoids and tonsils

<sup>1</sup> Discussion on Menigitis of Otitic Origin, *Proc. Roy. Soc. Med., Sec. Otol.* 1924, xxviii, 279. Joseph Breusmann, "Throat Infections in Children," *Arch. Ped.* March, 1925, xli, 145. Donald Paterson and G. W. Bray, "Tonsillar Hypertrophy and Infection as a Factor in Ill Health in Childhood," *Lancet* Nov. 21, 1925, ii, 104.

drain into the glands of the neck, and the tonsillar glands at the angle of the jaw on each side become enlarged when already the throat is septic. Where the glands have been repeatedly infected there is a tendency for the organisms to spread further down the chain or actually to reach the blood stream. After nose and throat infections various other organs are affected. These will be dealt with under Complications.

**Clinical picture and symptoms.**—The severity of nasal and throat infections varies enormously. There may be merely a slight running nose, or the child may develop a severe quincy or Vincent's angina. The temperature may be slight or high. If the mucous membrane of the nose and the adenoids are involved there tends to be headache. When the tonsils and pharynx are involved, there may be pain on swallowing, and general fever, lassitude and drowsiness. The whole infection may settle down in 24 hours or continue for weeks or months, depending on the organism and the type of child. With each infection the adenoids may swell causing nasal obstruction and snoring. Between attacks this may not completely subside, so that broken sleep and restlessness are features.

**Complications.**—The complications of upper respiratory infections are many and varied, and are of the utmost importance. *Laryngitis* and *bronchitis* commonly follow catarrh of the nose or throat. *Otitis media* is the direct result. *Umbilical colic* is often associated with sore throats. It is difficult to explain the aetiology of this, but it is probably due to enlarged mesenteric glands, caused either by swallowed infection which has passed through the intestinal wall to the glands, or by infection reaching the abdominal glands by way of the bloodstream. It is much more likely to be the former.

**Acidosis attacks**—Accompanying an acute sore throat or catarrh of the nose the child may vomit, probably due to sudden cloudy swelling of the liver cells (mild hepatitis). Such attacks are wrongly called acidosis attacks because of the presence of acetone in the urine. They are really 'chills on the liver' or old fashioned "bilious colics". They may be accompanied by constipation or, on the other hand, by diarrhoea. If the infections appear over and over again at regular intervals a wrong diagnosis of cyclical vomiting may be made (see p. 123).

**Rheumatism**—Rheumatism almost certainly gains entrance to the body by way of the tonsils.

**Nephritis**—The acute hæmorrhagic type of nephritis is most commonly the result of tonsillar infections.

**Stomatitis**—This may result from infections of the tonsils, especially with the spirochete of Vincent and the fusiform bacillus.

**Peritonsillar abscess**—This is uncommon before the age of five or six (see p. 125).

**Retropharyngeal abscess**—This is most common in the first few months of life.

Upper respiratory infections undoubtedly precipitate *asthmatic attacks* in allergic children.

In older children *antritis* may be directly traced to an infection from tonsils and adenoids.

**Treatment.**—In all cases the child is best in bed as there he is less likely to become chilled and can less easily convey his infection to others. The temperature of the room should be about 60° F and cross ventilation is desirable. Spraying the inflamed surface with liquid paraffin containing a drop or two of oil of eucalyptus to the ounce is recommended. Where the temperature is raised and the throat shows a moderate or severe degree of inflammation one of the Sulpha drugs should be given at once (For dosage, see p 333). In very mild infections a mixture of potassium chlorate and sodium sacchylate every four hours is most beneficial. For a child of three the mixture should be —

Pot chlor 2 grains  
Sacch sal 3 grains

Or, for a child of 5 years —

Acetylsacchylate acid 3 grains  
Sacch lactose 3 grains

One powder with a little water to be  
used as a gargle and swallowed

Painting the throat is distressing but where a follicular tonsillitis is present or the tonsils are much swollen one good painting with a weak tincture of iodine is often most effective. The bowels should be kept well open with grey powder at bed time and milk of magnesia or some saline such as *Frano's Fruit Salt* first thing in the morning. The diet should be that for a fever patient (see p 329).

**Operative treatment**—There is no more successful operation from the clinician's point of view than the removal of septic tonsils and adenoids in childhood. Children who have been ailing and pale with a tendency to earache or discharging ears with repeated feverish turns and gastric attacks become surprisingly well after tonsillectomy. The operation for choice is that of tonsillar dissection as taught and practised at Great Ormond Street. In this way the lingual portion of the tonsil is obtained as well as the faucial portion. Gullotining the tonsils except in the hands of a great expert may not be entirely successful as the lingual portion is often left behind. The operation has however, the advantage of requiring only a short anaesthetic.

**Anæsthesia**<sup>1</sup>—Paraldehyde by rectum as a premedication before anaesthesia has been a great success. The child goes to sleep some time before the operation and awakes some hours later with no knowledge of the procedure whatever. A drachm of paraldehyde is administered rectally for each 14 lb of the child's body weight about an hour before the operation. It is best given dissolved in ten times its volume of saline and 5 per cent glucose. Avertin<sup>2</sup> is a useful substitute. The following notes on basal anaesthesia are by Neil MacDonald senior anaesthetist at the Hospital for Sick Children Great Ormond Street. They are abstracted from *Abdominal Surgery in Childhood*, by L. E. Barrington Ward<sup>3</sup>. The basal

<sup>1</sup> H. S.ington, "Pre-medication by Paraldehyde in Children," *Proc Roy Soc Med (Sect Anæst.)*, 1928, xiii, 7.

<sup>2</sup> H. K. Ashworth, "The Use of Avertin for the Production of Basal Narcosis in Children," *Arch Dis Child*, No 63 June 1936, 4, 157.

<sup>3</sup> Oxford Medical Publications, 1937.

narcotic plays a real part in children's surgery, and allows a child to have an operation without being frightened by the anæsthetic

### Atropine

Infant	$2\frac{1}{2}$ grains
A few months old	$1\frac{1}{2}$ grain
A child of 3 years	$2\frac{1}{2}$ grain

is given hypodermically three quarters of an hour before operation. If given by mouth this dose should be  $\frac{1}{2}$  times that quoted

### Local Anæsthetics

**Nembutal**.—The dose is 0.6 grain per stone of body weight. Although supplied in a capsule it should be removed from the capsule and mixed with orange juice or honey or something sweet. It is given by mouth three quarters of an hour before the induction of anæsthesia, the child lying in a darkened room.

**Paraldehyde**. Sixty minims of paraldehyde per stone of body weight, dissolved in ten times its volume of normal saline is given slowly *per rectum*  $\frac{1}{2}$  hours before the operation.

**Avertin**.—One tenth of a cubic centimetre of avertin fluid per kil. gram of body weight in  $2\frac{1}{2}$  per cent. solution in distilled water is run in *per rectum* 20 to 30 minutes before the operation. It is probably best to run in only three quarters of the solution then await results for a few minutes and add the remainder if required. It acts much more quickly than paraldehyde but it is often not retained unless a previous rectal wash-out is given. The effect lasts several hours.

### Injection of Potassium Iodide (Compensated) (For rectal injection after tonsillectomy)

	Age 1 year	Age 2 years	Age 4 years
Potassium Iodide	10 gr	20 gr	25 gr
Aspirin	5 gr	10 gr	15 gr
Mucilage of Tragacanth	$\frac{1}{2}$ oz	$\frac{1}{2}$ oz	6 dr
Normal saline solution	$2\frac{1}{2}$ oz	3 oz	5 oz

**The function of the tonsil.**—Opponents of the removal of tonsils by operation ask—If tonsils are given us for some reason and if it is true that they act as a filter and prevent infection passing from the outside of the body into the bloodstream why should they be removed? From early infancy to the age of three four or five children are continually getting slight tonsillar infections. On each occasion there is a mild engorgement and enlargement of the tonsil and it is thought that antibodies are formed against various types of organisms conferring some degree of immunity. It is a slow process of vaccination against myriads of organisms and seems necessary to protect us in later life. Once this immunization has taken place the tonsils seem to have served their purpose since they become definitely infected themselves and act as a source of sepsis rather than an aid against it. Tonsils which have been removed show, on section, tuberculous infection in some cases while in others they are actually bags of pus. Septic tonsil may occur at almost any age but are probably most common between the ages of three and five.

### RETROPHARYNGEAL ABSCESS

This occurs most commonly in infants and children under the age of 2 years. There is usually a history of pharyngitis followed by an infection of the retropharyngeal glands. There is an interval usually of several days or longer when one of the retropharyngeal glands breaks down, causing an abscess in the lateral wall of the pharynx.

**Symptoms.**—There is usually some temperature, but occasionally this is very slight. Swallowing is increasingly difficult, the child tends to hold the head backward. The breathing is noisy and, since the pharynx is obstructed, mucus tends to drip from the mouth and nose. There is usually a swelling due to a gland at the angle of the jaw on the affected side. A careful examination of the pharynx reveals the swelling and, on palpation, fluctuation may be felt.

**Prognosis and treatment.**—Very often such abscesses drain away themselves, either being coughed up or swallowed. When discovered the abscess should be opened and suction applied. Relief is immediate, and recovery rapid. Fluids should be administered for dehydration. One of the Sulpha drugs is indicated particularly in the early stages.

**Differential diagnosis.**—Retropharyngeal abscess occurs in older children suffering from Pott's disease and tuberculous curies of the spine. (See p. 280.)

#### PERITONSILLAR ABSCESS (Quinsy)

This may occur in both infants and older children. The symptoms to some extent simulate those of retropharyngeal abscess. The inflammation commences deep in the tissues, usually behind the tonsil, pushing it forward. There is difficulty in swallowing, much pain and high fever. Pus is formed and must be evacuated by a surgeon, or the abscess will burst and drain spontaneously. Early administration of the Sulpha drugs may prevent pus formation and is indicated in all cases.

#### ACUTE AND CHRONIC SINUSITIS<sup>1</sup>

Every acute naso-pharyngeal infection is bound to spread to the nasal accessory sinuses, for they are lined by an extension of the mucous membrane of the nose. Usually, the inflammation of the sinuses subsides with general recovery, but, if an ostium becomes blocked, matter may accumulate under tension in the cavity and thus acute sinusitis may be initiated. More often, chronic sinusitis results from inadequate but not complete obstruction, or from severe infection even with an adequate ostium.

Frontal sinusitis is rare in childhood; acute ethmoiditis is occasionally met in young children, and may cause proptosis and external paralysis of the eyeball (see p. 391). Infections of the antrum are common in children of all ages.

**Acute antritis.**—Acute inflammation of the antrum may occur during a severe cold or other upper respiratory infection. The ostium being blocked, there is accumulation of pus under tension. The temperature is raised, there is severe headache and there may be pain localized over the affected antrum. An X-ray will show opacity of the sinus. The object of treatment is to induce shrinkage of the swollen nasal mucosa so that the ostium opens and drainage is established. Shrinking drops containing ephedrine are indicated, e.g., *Endrine*, *Rhinitol* or *Glucoderm*. Older children often prefer a benzadrine inhaler. Inhalation of menthol in hot water is very helpful. Failing relief by such measures, the antrum may be aspirated but it should not be washed through in the acute stage.

<sup>1</sup> James Crooks and A. S. Sney: "Accessory Nasal Sinusitis in Childhood, with a record of Bacteriological Examinations." *Arch. Dis. Child.* No. 68, Dec. 1926, p. 41.

*The Shrinking Drops for the Nostrils*

R. Gluco fedrin with I haemen le

Sig —One or two drops in each nostril morning and evening

Before putting the drops in the nose, the dropper should be thoroughly warmed  
Where the eyes are affected use Bencardis Eye Drops

**Chronic antritis**—The chronic condition is often the outcome of repeated colds which have failed to clear up completely, and may depend upon a large mass of adenoids interfering with the proper nasal air way. The child has a stuffy, 'catarrhal' nose, and postnasal discharge from sniffing. Such discharge in the throat causes chronic cough, and may lead to bronchitis, pulmonary fibrosis or bronchiectasis. Headaches are common and general debility is marked. Catarrhal otitis media may result from a spread of infection along the Eustachian tubes. An X ray will show either opacity of the antrum or thickening of its mucous membrane. In a proportion of cases the chronic antral infection is kept up by an allergic reaction of the nasal mucous membrane, as seen in hay fever, and rhinorrhoea. Treatment by desensitization may be highly successful in such cases (see Allergy p 162).

Treatment of mild catarrhal antritis is on general lines. Shrinking applications to the nose are valuable but a change to sea air is of the greatest help, although bathing is forbidden. Adenoids causing nasal obstruction should be removed. Failing improvement from these measures and in more severe cases antral lavage is frequently followed by most satisfactory results. Obviously, further upper respiratory infections may lead to inflammation of the antrum again and colds must be avoided as far as possible and treated strenuously when they do occur.

Washing out the antra or sinuses by Proetz displacement, as practised at the Hospital for Sick Children Great Ormond Street, is most beneficial in selected cases.

**CERVICAL ADENITIS<sup>1</sup>**

This is one of the commonest sequels to tonsillitis, and is a very unsatisfactory one to treat from the standpoint of the general practitioner. It may occur in epidemics, but cannot be said to be in any way a new disease.

**Clinical picture and diagnosis.**—After an acute naso-pharyngitis, usually due to the streptococcus, the tonsillar glands enlarge at the angle of the jaw, and are painful. Shortly after this, other glands in the neck may enlarge, the swellings being uni or bilateral. At the slightest movement of the head exquisite pain is felt. The tongue is furred, and the temperature high and inclined to swing, it will be down in the morning and up in the evening. This state of affairs may continue for two or three weeks or more, and in a school may assume epidemic proportions. Some cases are accompanied by otitis media or mastoid disease, and in other cases the glands may break down and form abscesses.

<sup>1</sup> R. Miller, "Epidemic Streptococcal Adenitis," *Brit. Med. Jour.*, Jan. 18 1930, I, 105. H. C. Cameron, *Clin. Jour.*, Jan. 1935, I, 2.





FIG. 20. Immobilization of head by application of cotton bandage over the cotton wool



FIG. 19. Splinting the head in cervical adenitis showing cotton wool wound round the neck

In the infant with otitis media suspicion may not fall on the ears merely from observation. The drums should be examined in every case. The child often shows toxæmia and may even behave as in meningitis, holding the head back and screaming at intervals. Very frequently there is an accompanying tonsillitis when the glands in the neck are enlarged. The baby's action in putting its hand to its ear cannot be taken as a sign of earache as a normal infant will often do the same thing.

**Prognosis and treatment.**—Earache in children should always be taken seriously, and a careful examination of the drums be made. If wax is present the ear should be syringed out with a Higginson's syringe using *tepid alkaline lotion* e.g. a teaspoonful of sodium bicarbonate in a tumblerful of lukewarm water. If the drums are red and early bulging is detected paracentesis should be undertaken. A golden rule for the practitioner is when in doubt perform paracentesis. A thin sero-purulent discharge will flow for the next two or three days or a week and later become scanty and thick finally drying up with healing of the tympanic membrane. If the drum is allowed to burst a small portion is destroyed and complete healing is not so common the discharge continuing very often for some weeks. Fomentations to the ears or a hot bottle under the head are very valuable if applied early when the earache first commences and continued until the discharge has ceased. With very slight earache, before redness can be detected drops of carbolic and glycerin should be instilled—

Carbolic acid 2½% in glycerin	1 of each
Distilled water	1 equal parts.

Or at care should be taken that the external auditory meatus is not allowed to become infected by the discharge from the ear, as an eczema may develop giving rise to a chronic discharge. To prevent this drops of rectified spirit are useful—

Boric acid	8 grains
Alcohol	120 minims
Water	120 minims

The directions for treatment at the Hospital for Sick Children are along these lines. The mother is given an all rubber rat tail syringe and this powder—

Sod borate	1 oz
Sod bicarbonate	1 oz
Sod chloride	2 oz

**Directions.**—One teaspoonful in ½ pint of boiled water.

Syringe out the ear with this lotion dry and until the 1. ric and alcohol drops, and lightly stopper the ear with cotton wool. Repeat two hourly if necessary.

With the advent of the Sulpha drugs the incidence of otitis media has been considerably reduced. If given in the early stages they appear to abort a considerable proportion of cases. (For dosage see p. 183)

**Acute mastoiditis.** **Ætiology.**—This condition often accompanies acute otitis media. In the infant its onset may be very rapid and within 24 hours the clinical picture may be complete. The infection usually spreads from the middle ear to the mastoid antrum infecting the mastoid cells. The organisms are the same as in acute otitis media.

**Clinical picture.**—The child becomes drowsy and lethargic, and

may vomit. The temperature is high accompanied by headache. Very often, with the onset of mastoiditis the earache ceases and the child may appear quite bright at intervals although generally drowsy. Careful examination may reveal a slight oedema over the mastoid process or the oedema may be marked with forward displacement of the ear. Occasionally however no oedema is present and there is little or no tenderness over the mastoid. Redness and swelling of the posterior wall of the external auditory meatus is a help in such cases and transillumination showing opacities in this region is of value.

**Treatment**—The treatment is surgical and should be considered an emergency operation: the mastoid being opened immediately and freely drained. The administration of sulphonamide at the earliest possible moment is indicated (see p. 383).

**Complications**—Occasionally infection of the lateral sinus meningitis and cerebral abscess occur.

**General remarks**—Prevention of otitis media and mastoid disease.—Undoubtedly early removal of infected tonsils and adenoids is by far the best prophylaxis. Certainly this should be done in every case in which there has been earache. Septic teeth should also have attention.

**Ear discharge (otorrhœa)**—The discharge may be from the external auditory meatus due to the presence of some foreign body or to a chronic eczema or from the middle ear due to an otitis media. The ear should be carefully syringed out with an all rubber rat tail syringe, using an alkaline lotion. Septic tonsils and adenoids should be removed in every case of chronic ear discharge due to middle ear infection as this is often the best and only means of doing away with the infection (see Otitis media p. 138).

**Serous meningitis (otitic meningitis)**<sup>1</sup>—During the course of an acute otitis media or mastoid infection signs of meningitis may occur. Headache, drowsiness and vomiting with or without neck rigidity, may be present. In such cases it is best to perform lumbar puncture, and, if the fluid shows an increase of cells and a raised albumin content, a paracentesis should be performed or an exploration of the mastoid is indicated. If this has already been done on one side it may need to be done on the other and a search for a further source of infection should be made in the region of the mastoids. Sometimes, plugging the lateral sinus or tying the internal jugular vein may be necessary.

## LOWER RESPIRATORY TRACT INFECTIONS

### ACUTE LARYNGO TRACHEO BRONCHITIS

Where there is marked inflammation and congestion of the larynx and trachea in addition to bronchitis a very serious condition sometimes arises with dyspnoea and much general upset. The child tends to suffocate from the thick secretion. The mortality is high about 50 per cent in cases reported by Brennemann<sup>2</sup> and others. Bronchoscopy, sucking clean the trachea and bronchi at intervals, or tracheotomy, appears to be

<sup>1</sup> Discussion on Meningitis of Otitic Origin, *Proc. Roy. Soc. Med. Sec. Child.* 1931 xxvi, 572.

<sup>2</sup> J. Brennemann et al., *Amer. Jour. Dis. Child.* April 1928 iv, 667.

the most efficient treatment. An oxygen tent is indicated, and one of the sulphonamide preparations (see p 383). Moist warm air is an essential

## BRONCHITIS

**Ætiology.**—A perfectly healthy child may develop acute inflammation of the trachea and bronchi as a result of a massive infection with some virulent organism such as the influenza bacillus. A less virulent organism may cause this when the child has been exposed to a chill. An ill, debilitated child is more likely to develop bronchitis, especially as an extension from an infection in the upper respiratory tract. The child with rickets and a catarrh of the naso-pharynx also tends to develop bronchitis by an extension of this infection. The same may be said of children with heart or kidney disease, in whom the circulation is not good. Bronchitis commonly follows the naso-pharyngitis which accompanies measles, whooping cough, chicken pox and scarlet fever. Some children develop what is termed bronchitis while they are teething, but it is a matter of doubt whether this actually is bronchitis or merely a cough due to an excess of saliva collecting around the larynx.

**Pathology.** *Physical signs and their interpretation*—It is necessary in the first place to define the terms used in describing physical signs. The author suggests the following:

*Rhonchi* are dry sound, either low or high pitched. There are so called whistling, sibilant and sonorous rhonchi and these are produced by the air whistling through the smaller bronchioles over portions of loose or tenacious adherent mucus or mucopus.

*Râles* or *crepitations* (these are synonymous) may be low or high pitched and are short, moist sounds, small or large in size, depending on their site. The smaller ones are undoubtedly produced by the moist walls of collapsed alveoli springing apart. The larger moist sounds may be produced by air passing through mucus in some of the smaller bronchioles. Crepitations of the smaller type, therefore, imply lung collapse and those of the larger type imply bronchiolitis. Large moist sounds are often referred to the chest from the larynx or trachea where they are produced, especially in a teething baby.

On examining the chest of an infant or young child with bronchitis the following physical signs may be found. There may be high or low whistling rhonchi as discerned with a stethoscope. Anyone standing near the child may hear wheezing breath, and the hand on the chest detects the same wheeze. If the smaller tubules or bronchioles are affected, and especially if the exudate is more profuse and thinner, moist sounds both small and large are present. The smaller bronchioles, becoming blocked with exudate, do not allow the air to reach the alveoli except at intervals, and minute areas of collapse are therefore present over the whole of the lung surface. With each breath some of the collapsed alveoli are inflated, giving rise to showers of fine crepitations. If low pitched, they suggest collapse, but if high pitched they suggest areas of consolidation or broncho-pneumonia, which convey the sounds easily to the ear.

Bronchitis is, as a rule, bilateral, that is, both lungs are involved.

and usually the distribution is diffuse. Very often simple pneumonitis<sup>1</sup> or mild broncho-pneumonia is present in cases wrongly diagnosed as acute bronchitis.

**Clinical picture**—It cannot be said that bronchitis has two definite consecutive stages, namely a dry stage when the surface is congested and inflamed and a second stage when there is much mucous secretion and moisture. With some infections the bronchioles seem to stream, just as the nose does from the beginning. With other infections there seems to be a period of dry inflammation during which the cough is short and hacking, and pain accompanies it, followed later by a period of moistness or looseness when much relief is obtained from coughing up the mucous or bronchial exudate. The temperature is almost invariably raised in bronchitis to 100° or 101°. The type of cough depends on the nature of the secretion. The respirations are increased. The cheeks are flushed, and the child is drowsy and irritable. Many children tend to be asthmatical, that is, along with an attack of simple bronchitis there tends to be an additional spasm of the bronchi and bronchioles giving rise to so called bronchial asthma. This is considered later under the heading Asthma (see p. 160). In such cases there are characteristic expiratory rhonchi, which are not common in simple bronchitis.

**Treatment.**—**Temperature of the room**—This is best kept at about 60° F. It is necessary to have a nursery thermometer. Warm, fresh air is absolutely essential and this may necessitate open windows. An open fire aids ventilation. It is wrong to have more than one adult in the sickroom at a time.

**Diet**—This should be the maximum that the child is able to digest. It is a great mistake, however, to feed small amounts often, unless food is being utterly refused. For the older child the fever diet mentioned on p. 329 should be resorted to, but the regular times of feeding should as far as possible be maintained. Between the feeds, and during the night, plain water, sugar water, barley water or diluted orange juice with sugar should be administered in large quantities. Much milk should not be given.

**Local treatment**—Where the cough is dry, hacking and unproductive, and examination of the chest shows high pitched rhonchi with a minimum of moist sounds, a cresolene lamp may be indicated.

**Drugs**—With the advent of the Sulpha drugs the outlook and treatment of bronchitis has been considerably simplified. A suitable Sulpha preparation should be administered at once in all cases. (For dosage, see p. 383.) Where for some reason a Sulpha preparation is not tolerated or is thought inadvisable the following treatment is suggested.

In small infants certainly up to the age of 18 months or 2 years cough mixtures should be avoided. Upset to the stomach is more likely than is any benefit. In an older child two types of cough mixture may be indicated, first an expectorant mixture such as

R. Tr. specac. 25 minims  
Glycer. 10 minims  
Ammon. carb. 1 grain  
Aq. dest. ad 60 minims

<sup>1</sup> A. Morton Gill. A simple pneumonia in Childhood. *Med. Rec. N. Y. Jour.*, Jan. 1927. XXXVII \*0

older child Just as there is no upper limit to the age when broncho-pneumonia may be present so there is no lower limit to the age when lobar pneumonia may be present It seems fair however to say that in the first two years of life broncho pneumonia is to be expected and from two years onwards lobar or broncho-pneumonia may occur

**Seasonal incidence**—Cases of pneumonia are admitted to hospital most commonly in January February and March

### BRONCHO PNEUMONIA

So called *primary* broncho-pneumonia—that is broncho-pneumonia in an absolutely normal child without a previous bronchitis—does occur This has been described as pneumonitis disseminated pneumonia and also as primary atypical pneumonia depending on whether the infection be due to the pneumococcus Pfeiffer's bacillus or to one of the virus infections

In *secondary* broncho-pneumonia there are as a rule several organisms chief among them being the pneumococcus the streptococcus the staphylococcus and Pfeiffer's bacillus

**Ætiology**—As a rule broncho pneumonia may be said to be an extension from an acute bronchitis the infection having transferred to the terminal alveoli and through the walls of the smaller bronchioles to the surrounding lung tissue This type of infection is especially common after measles and whooping cough both of which are accompanied by a severe catarrh of the respiratory mucous membrane With confinement indoors in a lowered state of health extension of infection to the lung substance is rapid In other debilitating diseases such as rickets and acute or chronic diarrhoea pulmonary catarrh is frequently present and in such children the catarrh easily extends so that broncho-pneumonia develops

**Pathology**—If the lung lesion has been present some hours the various areas seen under the microscope may be described in this way commencing from the bronchiole First there is a region where the alveoli show *consolidation* some containing polymorphonuclears lymphocytes endothelial cells coagulated serum and the debris of red cells The next area further from the bronchiole shows the process in an earlier stage Here the capillaries surrounding the alveoli are *congested* and beaded or ruptured in places and the red cells may be seen pouring out into the alveoli Polymorphonuclears and serum and desquamated endothelial cells are present Still farther out the alveoli show extreme *congestion* in their walls only Beyond this there is an area of *collapse* of the alveoli the walls lying in close apposition Still farther, the alveoli are distended with air and there may be well marked *emphysema* It is suggested that this area of *collapse* *congestion* *exudation* and *consolidation* travels out from the bronchiole wall like the circle of waves on a pond produced by a dropped stone The *emphysema* is produced when the remaining portion of the normal lung attempts to take on the respiratory function for the damaged portion The collapse is due to normal lung being caught between the emphysematous portion on the one hand and the damaged portion on the other

Plates 5 6 7—Radiograms showing the resolution of a case of lobar pneumonia untreated by chemotherapy, over a period of one week



Plate 5—Radiogram taken after the crisis

*(Plates 6 and 7, following show the further stages of resolution)*



Fig. 1—Normal bronchial tree in a case of slowly resolving pneumonia.



Fig. 2 Extreme degree of tubular bronchiectasis throughout whole of left lung

(By courtesy of Dr. Leonard Lindley)

Radiograms taken after lipiodol injection,

Plate 2.



or is due to the terminal bronchioles leading to the collapsed portion being blocked with exudate. Healing and resolution must obviously occur first in the portion next the bronchioles and extend onward, since this was the first portion affected.

**Clinical picture.**—Physical signs and symptoms.—As a rule a pulmonary catarrh or bronchitis has been present for a varying length of time possibly a few hours days or even weeks. The extension of this catarrh to the terminal alveoli and through the walls of the bronchioles is determined by the nature of the organism and the general condition of the child. A dull confinement had feeding all lower the vitality of the child and allow this extension. The temperature rises steadily from its previous height of  $99^{\circ}$  or  $99.5^{\circ}$  to  $100^{\circ}$  or  $101^{\circ}$ . The cough which has been previously present, becomes much more marked and may be almost continuous. There is an expiratory grunt, and the normal rhythm of breathing is reversed so that the pause which usually takes place after expiration now takes place after inspiration. The pulse is rapid and instead of the normal ratio between respirations and pulse remaining as 1:4, it may be as 1:2. The rate of the respirations increases from the normal 20 or 25 per minute to 40 or even 60 per minute. The face is flushed and cyanosed the alae nasi work with each respiration the diaphragm and intercostals overact. The child is restless rolling his head from side to side on the pillow. The mouth is dry and the tongue furred. The lips are parched and the child is drowsy.

**Examination of the chest.**—In examination at the very outset may show the signs of bronchitis only harsh breathing inspiratory rhonchi and occasional low pitched moist sounds. In this type of pneumonia, as a rule, both lungs are affected. Once the disease is established, the child's breath sounds remain harsh but they are higher in note. Where small areas of broncho pneumonia have become confluent, there may be tubular or bronchial breathing. Accompanying the breath sounds are many small moist crepitations which are high pitched and crackling, indicating areas of consolidation. The percussion note may be resonant or even hyperresonant (due to compensatory emphysema) or may be impaired. The vocal resonance and vocal fremitus are both increased. During the course of the disease it is common for the microscopic pneumonic patches to coalesce, so that large areas, varying from the size of a shufin, to a whole lobe, may give the physical signs of consolidation. In such circumstances it is difficult to differentiate clinically between bronchio- and lobar pneumonia.

**X-rays.**—The physician will be surprised at the contrast between the physical signs as found by himself by percussion and auscultation and the picture revealed by a good X-ray. The stethoscope in the clearest hands may be totally misleading, and an X-ray may show widespread disseminated infection in the lungs or patchy consolidation where none had been previously suspected. (See Plate 4.)

It is well therefore not to believe that pneumonia is excluded by physical examination only and where there is ground for suspecting it, an X-ray of the chest should in all cases be taken.

Generally speaking X rays will divide pneumonias into the *bronchopneumonic* type with the lesions wide-spread and the *lobar* type where the lesion is confined to one or more lobes. Very often in the latter however other parts of the lungs are affected and the division is an X ray classification rather than a clinical or pathological entity.

**Complications and prognosis**—Should the right side of the heart overfill with marked congestion and enlargement of the liver and cyanosis the outlook is poor. Prognosis is bad also when diarrhoea accompanies pneumonia. In infants symptomatic diarrhoea is not uncommon. Convulsions due to cerebral congestion and toxæmia are a serious complication.

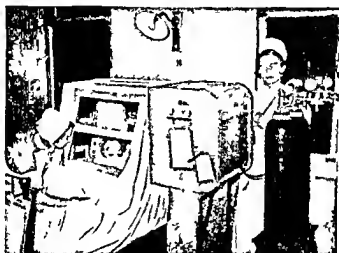


Fig. 21. A modern type of oxygen tent.

*Abdominal distension* accompanied by constipation further impedes respiration and requires purges or colicivage. *Empyema* is present in a proportion of cases (see p. 152). Another life complication is *bronchiectasis* which sometimes occurs, especially when the bronchopneumonia is streptococcal (see p. 155).

The prognosis depends on several factors but probably the most important is the age of the child. The younger the child the worse is the prognosis on the whole. The Registrar General's Report for 1937 shows the following figures:

PERCENTAGE OF DEATHS FROM PNEUMONIA IN ALL CHILDREN  
UNDER 15 YEARS OF AGE

Under 15 years Deaths	%	0-1 Deaths	%	1-2 Deaths	%	3-10 Deaths	%	10-15 Deaths	%
9,319	16.7	5,582	15.9	3,075	27.4	443	8.1	109	5.3

changing, and general attention should be arranged at as long intervals as possible, so as to ensure the maximum of sleep.

*Poultices*—With the advent of the Sulpha drugs this treatment is no longer necessary.

*Steam tent*—This is dangerous and should not be used.

*Tepid sponging*—During the hyperpyrexia, sponging the face and limbs with tepid water affords some relief.

*Serum*—As a rule, serum is not of use in broncho pneumonia. The bacterial flora is usually so mixed, containing several organisms, that no one serum could be said to be indicated.

*Sulpha drugs*—In all cases where a pneumonia is suspected and the temperature is raised, or where a pneumonia has been clinically or radiologically diagnosed, one of the Sulpha drugs should be given immediately (For dosage, see p. 383).

#### LOBAR PNEUMONIA\*

**Ætiology**—Of recent years the pneumococci causing lobar pneumonia have been divided into four main groups.

*Type I*—This is the mildest type, and is responsible for from 20–40 per cent of all cases, according to various statistics. The mortality is lowest but it causes the highest proportion of empyemata.

*Type II* is responsible for from 10–20 per cent of cases. This pneumonia is more severe, and there is definitely more marked toxæmia. The mortality is higher.<sup>2</sup>

*Type III* represents from 3–15 per cent of the total cases. The mortality is high, and the clinical picture that of a severe toxic infection.

*Types IV–XXIII*—This last large group has been further broken up and it is now claimed that great variations in the clinical picture occur. It represents from 30–40 per cent of the total incidence. The mortality is not severe.

In collecting sputum for typing from an infant or young child the author has found swabs, taken after the child retches, both useless and disturbing. He suggests that a fine rubber catheter be attached to a short glass cannula, while on the other end a further piece of rubber tubing is placed. The whole is now sterilized and the tip of the catheter is slipped over the back of the child's tongue. At once the sputum wells up from the trachea and can be sucked into the catheter and blown out into a sterile test tube. This may either be typed by the direct method, or injected into a mouse overnight and typed the following day.

**Site of origin and mode of spread.**—Experimentally, the infection takes place only when certain strains of pneumococci reach the smaller bronchi. This may occur naturally, when small droplets of infected material, such as sputum, are deeply inhaled and come to rest in these bronchi near the root of the lung. The infection then spreads through the walls to the alveoli. In man, acute lobar pneumonia originating near the root of the lung, spreads with a sharply defined margin, outwards and downwards, in the lower lobe or, on the right side, directly

\* D. T. Davies, H. O. Hodgson and L. Noel Whitty. \* A Study of Pneumococcal Pneumonia. *Lancet* April 1935, I, 791–819.

<sup>2</sup> D. Ley, "Further experiences of Serum in the Treatment of Pneumonia, *Lancet*, July 13 1935, II, 67.

in childhood does the patient appear so ill, and yet so seldom die. When he seems as bad as possible he suddenly recovers in a dramatic fashion. Complications such as empyema, fibroid lung and pericarditis, affect both the immediate and the ultimate prognosis for the worse. A leucocyte count of 20 000-30 000 is of good prognostic value. This should tend to fall along with the temperature. If it does not do so it suggests some complication.

**Treatment**—Since the mortality is extremely low in lobar pneumonia and if it is given careful nursing almost all uncomplicated cases over the age of two recover, there is little call for heroic measures. The advent of the Sulpha drugs has greatly improved the prognosis and treatment (for dosage see p. 993). As a rule the temperature shows some improvement within 48 hours of an adequate dose. Physical signs however lag greatly behind the clinical picture. Fresh air, careful feeding, occasional oxygen, supervision of sleep, tepid sponging, when necessary, and careful watching for complications are all that is required. (For details see Broncho-pneumonia p. 149.)

### PRIMARY ATYPICAL PNEUMONIA\*

This pulmonary condition has been described as pneumonitis (Gill), disseminated focal pneumonia (Seadding) and as atypical pneumonia by many other observers.

**Clinical picture**—The onset is gradual with an incubation period of 7 to 21 days. There is a dry paroxysmal cough with fever, headache, malaise and chills. There are few physical signs in the chest but an X-ray shows an increase of root shadows extending out from the hilum on one or both sides. There is usually a leucopenia and a normal differential count. Cold agglutinins in high titre are developed in atypical pneumonia for human red blood corpuscles of Group O.

**Ætiology**—There is no response to chemotherapy and the causal organism is generally thought to be a virus although this has not been sufficiently confirmed.

**Course**—This is mild and lasts two or three weeks or longer.

**Prognosis and complications**—It is rarely fatal. Among the complications are depression, mild encephalitis, meningismus, thrombophlebitis of limbs and pulmonary infarction.

**Treatment**—This is symptomatic.

### EMPYEMA

This is a purulent effusion into the pleural cavity, as a rule following pneumonia.

**Ætiology**—In the vast proportion of cases the organism present in both the primary pneumonia and the secondary empyema is the pneumococcus but in a certain number of cases the streptococcus, staphylococcus

\* Editorial "Primary Atypical Pneumonitis" *The Lancet* June 19th, 1913 II. 79. Editorial "Primary Atypical Pneumonia" *Brit. Med. Jour.*, Feb. 6th, 1913 I. 223. A. M. Gill, "Pneumonitis, *Brit. Med. Jour.* March, 1938 I, 401. J. Seadding "Disseminated Focal Pneumonia" *Brit. Med. Jour.* Nov. 13th, 1937 II. 50.

Pfeiffer's bacillus, and tubercle bacillus may be found. It is possible that, accompanying the primary pneumococcal infection some of the organisms settle in the lung and give rise to lobar pneumonia while coincident with this there is a purulent pleurisy. In some cases however it seems clear that there has been an extension of the infection from the lung to the pleura at an interval of some days after the actual onset of the pneumonia.

**Pathology**—The thin almost colourless effusion rapidly fills the pleural cavity. Quickly it becomes milky opaque and later purulent. Within a few hours a thick layer of lelted lymph coats the parietal and visceral pleura. If this is opened and drained large clotted lumps are evacuated. There is a tendency for the pleura whether drained or not to be permanently thickened over the surface of the lung and this thickening tends to extend into the lung substance giving rise to interstitial pneumonia (see p 155). Often the two layers of pleura are bound together after an empyema or strong adhesions are formed.

**Clinical picture**—There are two common types of empyema. In both the clinical picture is much the same. In the first (*metapneumonic*), which is much the more common the child has what appears to be an ordinary lobar pneumonia. The temperature settles and he appears to be doing quite well. Three or four days later the temperature rises slightly and he does not seem so well. Each day the temperature is slightly higher until it may reach  $102^{\circ}$  or even  $103^{\circ}$ . X rays show that the pleural cavity contains fluid. The other type (*sympneumonic*) is less common and in this the temperature shows no inclination to settle down at the end of a week or 10 days, although an ordinary pneumonia has been diagnosed. After having continued high for some days or longer the physical signs becoming more those of fluid than of pneumonia an investigation is made and pus is found.

In the first type it is probable that the pleural cavity was infected secondarily to the lung whereas in the second type it is considered probable that the pleural cavity and the lung condition were coincident and part of a general pneumococcal infection.

The prognosis in these two types is quite different and they must be considered separately. It is clear that early treatment with one of the Sulpha drugs will greatly alter both the clinical picture and the course.

Among the symptoms are pain in the affected part of the chest, a short hacking unproductive cough, and dyspnoea and cyanosis getting progressively more marked.

On examination the breathing and pulse-rate are found to be extremely rapid. The child is cyanosed, and the heart is displaced to the side opposite to that containing the empyema. On placing the hands over the two sides of the chest a definite thickening and oedema of the chest wall is detected on the affected side. On percussion, the note is stone dull either over the whole lung or over the lower part where the fluid has collected. On auscultation, the breath-sounds are either absent or much reduced. At the upper level of the fluid there is egophony. Vocal resonance and vocal fremitus are decreased. If the heart has been embarrassed the liver will be found enlarged.

**Course of the disease.**—Should the pus not be evacuated, the fever continues for some weeks or months. The pus may gradually point at or somewhere about the nipple or in the axilla, becoming a so called "empyema necessitatis." On the other hand in pure pneumococcal infections, the pus may be gradually reabsorbed leaving greatly thickened and adherent pleura. In this type there is a tendency for interstitial pneumonia to develop giving rise finally to a fibroid lung with bronchiectasis. This type is however uncommon.

**Prognosis.**—The prognosis very largely depends on

1 The age of the child. As in bronchio pneumonia in the very young child the prognosis is extremely bad. Before chemotherapy there was a mortality of at least 50 per cent in children under a year old but this diminished rapidly up to the age of two, when the mortality was comparatively small.

2 The previous health and general physical condition of the child. In rachitic or under nourished children, or those children who have some organic disease elsewhere the chances of recovery are greatly lessened.

3 The nature of the organism. In pneumococcal empyemata the prognosis is best whereas in streptococcal, staphylococcal or Pfeiffer empyemata the outlook is much graver.

**Treatment.**—All cases should be given the benefit of chemotherapy in the early stages (see p. 353). It is unlikely that it will be effective, however in the later stages once the pus is loculated. Even if pus is suspected and found during a pneumonia thorough drainage is still undesirable. A needle may be put in from time to time and the greater part can be evacuated by means of a syringe but the pneumonia should be allowed to clear up and the pus then dealt with by resection of a rib or the introduction of a tube between the ribs. Where the empyema is merely part of a general septicæmia much the best course is to wait until it shows signs of loculation or subsidence and it may be necessary to wait some days. The introduction of a needle and the aspiration of pus from time to time is called for if there is cardiac embarrassment. If pus is definitely detected and a decision has been made to evacuate it, resection of a rib or intercostal drainage is much to be preferred. When a rib has been resected and an extensive opening made in the chest wall, a finger may be inserted breaking down adhesions between the layers of pleura and evacuating large clotted lumps. This tends to prevent permanent adhesions, thickening of the pleura and interstitial pneumonia. The tube should be left for some days until no discharge appears, as there is a great tendency to too-early closure. Intercostal drainage by the de Pezzer self retaining drainage tube combined with washing out the pleura daily with some mild antiseptic, such as Fusol or Dakin's solution, has been successfully carried out of recent years in hospital practice. In this way osteomyelitis of the ends of the ribs, so likely after rib resection, is avoided. Once the pleural cavity has been closed, exercises are encouraged, such as blowing into a Wolfe's bottle, inflating toy balloons, blowing on whistles and horns, these tend to cause full expansion of the lung. Sea air shortens convalescence and improves the child's general health. Cod liver oil is indicated. (For treatment with penicillin, see p. 395.)

**Complications**—Accompanying an empyema and producing a fatal result the following have been found: pyopericardium, suppurative meningitis, lateral sinus thrombosis and gangrene of the lung. After operation, in some cases, there is still a high temperature and persistent toxæmia. Late in the course of the disease severe diarrhoea is often a complication, in such cases there is probably an accompanying septicæmia. The author has found blood transfusion most useful.

### LUNG ABSCESS

In the British Isles lung abscess is comparatively rare. In America the incidence appears to be much higher.

**Ætiology.**—The commonest cause appears to be the aspiration of small portions of adenoid tissue or infected blood after the operation for removal of tonsils and adenoids. It may occur, however, along with a bronchio pneumonia or any respiratory infection, or after aspiration of any foreign material.

**Clinical picture.**—Usually three or four days or later, after the operation for tonsils and adenoids the child develops a short cough and appears less well. The temperature rises. An examination of the chest reveals an area of poor air entry with many fine crepitations. An X ray will show an opaque area at first and later actual cavity formation.

**Course.**—After a varying time, usually between one and three weeks the child, during a fit of coughing, may bring up some thick, foul smelling material, and it will be found that the abscess has drained into a bronchus with immediate relief and recovery. Rarely, an empyema forms.

**Treatment.**—Usually no specific treatment is necessary beyond nursing care, as described under pneumonia. In the early stages a course of one of the Sulpha drugs should be given.

Surgical treatment is occasionally necessary and may be attempted after a bronchoscopy, or after fixing the two layers of pleura and draining externally. The prognosis in lung abscess in childhood is good on the whole, and few sequelæ occur as a rule. Occasionally, however, fibroid lung results and this in turn may necessitate lobectomy.

### BRONCHIECTASIS

(BRONCHIOLECTASIS, FIBROID LUNG, INTERSTITIAL PNEUMONIA)

**Ætiology.**—*Fibroid lung, or bronchiectasis is most commonly the sequel of unresolved pneumonia, the pneumonia being as a rule of the broncho-pneumonic type, following on measles or whooping cough. The streptococcus is the organism most commonly concerned. In little children the tubercle bacillus is seldom the primary cause of the condition but later it may attack the damaged lung. Fibrosis of the lung arising from the pleural surface and passing inward by the septa may follow an undetected empyema. Foreign bodies in the bronchus and enlarged glands pressing on the bronchioles may cause retained pulmonary secretions with a low grade pneumonia or collapse and, finally, bronchiectasis. There is no doubt that a definite association exists between antritis, other*

sinusitis and pulmonary infection such as bronchiectasis, this must therefore be sought.

**Pathology.**—With the ordinary pneumococcal infection the interstitial tissue of the lung is less affected, the brunt of the infection falling on the alveoli. In streptococcal infections the inflammatory reaction in the interstitial tissue is great with consequent scar tissue formation and contractures. If the inflammation is slow and long continued fibrous tissue is invariably formed, with permanent damage and scar tissue. The tubercle bacillus, the spirilla, or some less virulent and lower grade organisms may also cause fibrous tissue formation (Fig. 24). Probably the commonest sequence of events, however, is for collapse of a lobe to occur as the result of broncho pneumonia. Bronchiectasis then follows as the consequence of long retained bronchial secretions weakening



Fig. 24.—State of lung in bronchiectasis.

the walls of the bronchi. Inhalation of a foreign body or pulmonary tuberculosis should, however, not be lost sight of as a cause of collapse and consequent bronchiectasis. Congenital cystic disease of the lung or congenital atelectasis, will both give rise to bronchiectasis in later life.

**Symptoms and diagnosis.**—There may be a complaint that since a broncho pneumonia after measles or whooping cough the child has never been well. An examination will show bronchiectasis. On the other hand in some cases no history is obtainable, the process having apparently come on slowly and insidiously. A severe cough is always a feature. It may be barking and harsh or hoarse, suggesting enlarged mediastinal glands or a foreign body. Often it is accompanied by an inspiratory whoop, very suggestive of whooping cough. The breathing is more rapid than normal, and on exertion the child may become cyanosed and

<sup>1</sup>R. W. B. Ellis: "Aetiology of Bronchiectasis in Childhood," *Arch. Dis. Child.* 1923, vol. 3, p. 11. H. H. Mott, "Clinical and Pathological Study of Bronchiectasis," *Quart. Jour. Med. New Series*, No. 3, July 1921, p. 457.



dyspnoea. There may be *clubbing of the fingers* in cases of long standing. Probably one of the first things to be noticed is the *odour of the breath*, which becomes fetid and offensive. The *sputum* is characteristic, containing much pus, and may amount to an ounce or more in the day. The paroxysms of coughing are not frequent, perhaps three or four in 24 hours but as a rule, these are productive, if the child is in a suitable position to ensure drainage of the cavities. *Examination of the chest* often reveals definite flattening or retraction of the ribs on the affected side. The left lower lobe is much more commonly affected than the right—possibly there is some connection between the pressure of the heart on the left bronchus and bronchiectasis. On *percussion* the note is dull over the affected lobe. It may be so dull as to suggest fluid but it is usually what may be termed apple dullness not stony dullness. On *auscultation* the physical signs vary enormously, depending on whether or not the tubules contain fluid. If they are well cleared out the breath sounds are tubular or amphoric



Fig. 23 Clubbing of fingers and toes in a case of bronchiectasis

suggesting cavity formation. In addition the accompanying sounds are high pitched, large and splashing. If the tubules are partially filled, the breath sounds may be markedly diminished and distant, with a few accompanying moist sounds only. If quite filled as they are at certain times in the day, the breath sounds may be absent and there may be no added sounds. By inverting the child, so that the secretions tend to drain away, coughing is provoked and the cavities emptied, and in a few moments the auscultatory signs are completely changed. The question whether fluid is present in the pleura will often arise. It should be noted that in bronchiectasis the heart is drawn over to the affected side, and not pushed away from it as with fluid in the pleural cavity. The chest wall is also definitely flattened on the affected side. An X ray of the chest shows displacement of the trachea and other contents of the mediastinum to the affected side. The affected lobe of the lung casts a shadow, which may in some cases be definite enough to suggest empyema.

The question continually arises. Is this pulmonary lesion tuberculous or non tuberculous? A careful examination of the sputum will help, but, on the other hand, it should be realized that a non tuberculous fibrosis

may at any time become infected with the tubercle bacillus. In children a tuberculous pulmonary fibrosis tends to be progressive or to become miliary and end fatally. X rays and examination of the antra are necessary in every case of bronchiectasis.

**Bronchoscopy**—This is often of great value in ascertaining the reason why a lobe is collapsed and becoming bronchiectatic. During bronchoscopy a foreign body or gland occluding the lumen of a bronchus may be detected and removed, causing the re-expansion of the collapsed lobe. Lipiodol may be given to obtain a bronchogram during bronchoscopy.

**Lipiodol**—The modern investigation of bronchiectatic cavities by means of lipiodol has thrown much light on the whole condition. (Plate 8.) The procedure, however, is not without danger, and should on no account be undertaken by anyone who is not skilled in its technique.

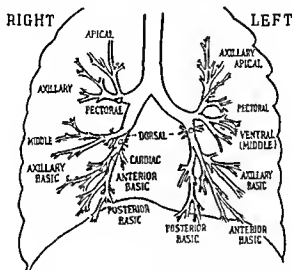


Fig. 26.—Diagram of the anatomical relation of the bronchi prepared by the late Mr H. P. Nelson. (It produced from the *Prison Hospital Reports*.)

General anaesthesia is obtained, with ether, gas and oxygen, avertin or nembutal given by the mouth or rectally or by paraldehyde rectally. A curved needle is passed through the space between the thyroid and cricoid cartilage and from 10–12 c.c. of lipiodol are slowly injected. This oily fluid runs down the trachea and enters the bronchus which is the most dependent. The child can be rotated to the other side, and more injected so as to reach that lower lobe and an X ray is then rapidly taken. If the bronchiectatic cavities are properly emptied, a perfect picture outlining these cavities will be obtained. (Fig. 26.)

Another method is to pass a catheter directly down the larynx and inject the lipiodol through the catheter. In older children the injection can sometimes be made without a general anaesthetic, by painting the back of the

throat with a solution of cocaine and then passing a fine catheter directly into the upper end of the larynx, or by grasping the tongue firmly with a piece of lint and drawing it forward, when the lipiodol can be trickled over the back of the tongue directly into the larynx. The posture of the patient, once the lipiodol has been introduced into the trachea is of the utmost importance in obtaining a good result.

X-rays give a fairly satisfactory picture of bronchiectasis even without lipiodol. But nevertheless it is possible to miss an atelectatic lung with bronchiectasis if the plate be slightly over exposed especially in the left lower lobe, which is hidden by the cardiac shadow. An under exposed soft film shows it best.

**Prognosis.**—A slight pulmonary fibrosis is quite compatible with good health and normal longevity, in fact after a few months of treatment no physical signs are given, nor can a pulmonary lesion be detected by X rays, the chest appearing quite normal. If however the child's health falls below par for any reason, recurrent bronchitis or pneumonia is apt to occur, and the fibrosis may progress to bronchiolectasis or bronchiectasis. In time, in severe cases, cardiac failure may follow with marked clubbing of the fingers, increasing cyanosis and dilatation of the heart. Much displacement of the heart, due to adhesions in the pleura dragging over the mediastinal contents, may largely account for this. Occasionally in the most severe cases where the sputum is fetid and offensive cerebral abscess occurs. In some cases hæmoptysis, especially when there is large cavity formation, may prove fatal. In untreated cases the mortality in children is approximately 50 per cent in a ten year period. Slight cases accompanying atelectasis appear to recover completely. Bronchiectasis is however, a progressive disease in childhood.

**Treatment.**—Each case should be treated on lines similar to the modern method of treating pulmonary tuberculosis. Fresh air is essential. An open air life should be arranged and continued for long periods. The diet should be well balanced and nourishing and cod liver oil is indicated.

The following simple cough mixture from the Great Ormond Street pharmacopœia is useful, and can be given four or five times daily.

R. Tinct. benzoin co. 2½ minims  
Tinct. opii co. 5 minims  
Creosote, ½ minim  
Syr. tolu. 5 minims  
Mucil. acac. 10 minims  
Aq. menth. pip. ad 60 minims

As an alternative the author suggests vapor iod. ethereus (B.P.C.)

Ethereal sol. of iodine 120 minims  
Icohol 120 minims  
Creosote, 60 minims  
Alcohol 90 per cent, ad 1 ounce  
Ten minims to be used in an inhaler

The best medicinal treatment is inhalation with a Burney Yeo mask, 2 or 3 drops of a mixture of creosote, 1 part, and chloroform, 2 parts, should be given at intervals of three or four hours.

The author is fortunate in being associated with a surgical colleague who was a pioneer in the treatment of bronchiectasis.

**Posture**—In severe cases of bronchiectasis the children lie across a double inclined plane in bed, for from 8 to 12 hours per day, the head lying from 12 to 16 inches lower than the buttocks. The cavities empty readily and the sputum which may be 50 or 60 c cm at first lessens gradually to from 5 to 10 c cm. It is noticed that a swinging temperature becomes normal and many pounds are gained in weight during the four to six weeks spent in this position.

**Surgical treatment**—After lobectomy<sup>1</sup> which is extremely well tolerated the child seems to regain great health and vigour. In 88 cases between four and sixteen years of age operated upon for lobectomy by Tudor Edwards there was no mortality. There is no doubt in the author's mind that this is the treatment of the future in certain selected cases.

### ASTHMA

**Ætiology** Age—Asthma may occur at any age and in some cases appears to commence almost from birth. Two thirds of the cases occurring before puberty commence before the age of 3 years.

**Sex**—There seems to be a proportion of at least two boys to one girl.

**Heredity**<sup>2</sup>—In more than half the cases there is a family history of asthma, hay fever, eczema or urticaria. Migraine and tram and bus sickness have also a familial tendency. This suggests a marked tendency for asthma to be inherited or at least for an instability of the nerve supply of the bronchioles to be hereditary.

**Cause of the symptoms**—It is thought that asthma is (1) a spasm of the bronchial muscles (2) an irritation and swelling of the lining mucous membrane of the bronchioles or (3) obstruction of the smallest bronchioles by plugs of mucus. It is possible that all three factors operate in some cases.

**Exciting factors**—Various factors may precipitate an attack of asthma. Among the commonest are (1) infections especially of the nasopharynx (2) hypersensitiveness to foods and/or inhalants.<sup>3</sup>

**1 Infections**—Nasopharyngeal infections together with the virus of the common cold or influenza apparently precipitate an attack of asthma in an allergic child. The mechanism of this is not clear but it appears that the mucous membrane of the nose, throat or respiratory tract in its inflamed state allows inhalants to which the child is allergic, such as dust or particles of feathers, to come into intimate contact with the tissues, resulting in a spasm of the bronchioles.

**2 Allergy** (Hypersensitiveness to foods or inhalants)—Various foods for example egg white or egg yolk or fish, may produce an attack of asthma if given to a child who is highly allergic to such substances. More commonly however inhalants such as feathers, horse hair, house dust, various pollens, cat or dog fur, orris root are the exciting factors. This may account for nocturnal attacks as the child is in intimate contact

<sup>1</sup> A. Tudor Edwards. Treatment of Bronchiectasis. *Brit. Med. Jour.* April 27, 1939, 1, 809.

<sup>2</sup> C. W. Bray. The Hereditary Factor in Asthma and Other Allergies. *Brit. Med. Jour.* Mar. 1, 1930, 1, 384.

<sup>3</sup> C. W. Bray. Recent Advances in Allergy. (Churchill) 3rd ed. 1937.

with his feather pillow, eiderdown and horsehair mattress Train journeys in dusty carriages dancing classes with powdered floors (orris root) and picnicking in the country among the pollens often produce an attack of asthma attributed to over excitement or over eating rather than to its true cause

**Clinical picture**—The child may be put to bed with what appears to be a slight cold with or without a temperature but wakes during the night much distressed coughing and wheezing and unable to get his breath There is cyanosis and dyspnoea and he looks anxious and careworn and complains of tightness about the chest The attack may be over in a few hours or cease on waking in the morning on the other hand if accompanied by a fever it may continue for some days and manifest some degree of true bronchitis

**Classification**—The term *spasmodic asthma* may be reserved for cases starting very acutely and accompanied by extreme cyanosis and respiratory distress In *bronchial asthma* on the other hand the spasm is less marked with less distress but as a rule there is an accompanying or superadded bronchitis with mild fever The author believes that there is no clear cut type of asthma the attacks being sometimes short and sharp and sometimes long drawn out lasting several days The individual does not consistently suffer from the same type of attack and is sometimes febrile and at other times afebrile

**Physical signs**—The percussion note is unchanged or hyper resonant On auscultation both inspiratory and expiratory rhonchi are heard The excursion of the chest is small and the air entry extremely poor The chest may be barrel shaped In asthma the dyspnoea is primarily expiratory

**Diagnosis**—In every case organic pulmonary disease must be carefully excluded as asthma may be simulated by such conditions as fibroid lung or bronchiectasis especially cases of long standing An X ray of the chest should be undertaken whenever possible and an intracutaneous tuberculin (Mantoux) test is of value as a routine Repeated attacks of bronchitis for no apparent cause in a child should always suggest asthma The blood count showing an eosinophilia of from 4 per cent to 10 per cent or even 20 per cent strongly suggests asthma so also does a family history Asthma should also be suspected if the child has a previous history of well marked eczema or urticaria

**Preventive treatment.**—Where an attack commences with a fever it is reasonable to suspect an infection and a careful examination of the tonsils and ears should be made where necessary infected tonsils and adenoids should be removed In some cases small inadequate noses will be found and such cases do extremely badly until puberty when the nose appears to enlarge which gives relief

When the child is sensitive to feathers or horsehair both of which are commonly found in bedding it is advisable to substitute a flock kapok or rubber pillow and mattress and to use blankets instead of an eiderdown When he is sensitive to house dust, carpets and curtains should be reduced to a minimum and a vacuum cleaner should be used for cleaning

The skin protein tests may show a sensitivity to one of the foods, the commonest of which are egg yolk and white lactalbumin of milk, fish, cereals and fruit

*Eggs* should be hard boiled, for 5 to 10 minutes

*Milk* should be boiled, cooled and the skin removed from the top

*Fish* should be steamed or boiled

*Cereals or fruit* should not be eaten raw

The following hydrochloric acid mixture may be given with benefit

R Acid hydrochloric dil 10 minims  
 Pulv dextrose 15 grains  
 Aqua ad 60 minims

Sig — 120 minims three times daily in orange juice or water immediately before or with meals over a prolonged period

It is often found that a routine dose of chlor of ephedrine or half a grain of ephedrine hydrochloride, one teaspoonful given at bed time, and perhaps repeated once in the night will prevent attacks of acute spasmodic asthma

**Treatment of an attack.**—Where an infection such as tonsillitis or otitis media is responsible for an attack with resulting fever one of the Sulpha drugs should be given at once (for dosage see p 388). In the very severe spasmodic case one or two minims of adrenalin may be given subcutaneously and repeated if necessary. One half grain of ephedrine hydrochloride, or one teaspoonful of chlor of ephedrine will be found effective in the sub acute case and may be repeated once or twice in the night if necessary. In all cases where the child is restless and wakeful a suitable dose of phenobarbitonum (B P) should be given (see p 397)

**Method of skin-testing.**—Sensitivity to foods is best ascertained by the scratch test method. A series of scratches is made on the inner surface of the forearm with a scarifier care being taken that blood is not drawn. Solutions of groups of foods such as man's vegetables, fruits, are applied to the scratch and the result can be read as a rule within 10 to 15 minutes. A positive reaction is revealed by a reddening of the skin with a central wheal at the site of the scratch. Sensitivity to inhalants is best ascertained by a series of intra cutaneous injections of group inhalants such as feathers hairs and pollens. A positive skin reaction is revealed by a white wheal surrounded by a reddened area, appearing within five or ten minutes. In both scratch and intra cutaneous testing control tests are necessary

#### Method of Desensitization of Asthmatics with Mixed Inhalant Solutions.\*—

The solution for subcutaneous injections is issued in three bottles as follows —

- 1 Ordinary course solution diluted 1 in 4 with sterile water (Blue label)
- 2 Ordinary course solution (Black label)
- 3 Continuation course (five times as strong as 2) (Red label)

The treatment is given as follows —

- 1st day 1 minim of the further diluted 'ordinary course solution' (bottle No 1) followed either later that day or the next day, by 3 minims of the same solution

\* An outfit is supplied by Messrs. Benard Ltd. Gorgate Hall, Dereham, Norfolk.

2nd day	1 minim of the "ordinary course solution" (bottle No. 2) in the morning 3 minims at bed time
3rd day	5 minims of the ordinary course solution (bottle No. 2) in the morning 7 minims at bed time
4th day	9 minims of this solution in the morning (bottle No. 2) 12 minims at bed time
5th day	7 minims of the continuation course solution (bottle No. 3) in the morning 6 minims at bed time
6th day	8 minims of this solution (bottle No. 3) in the morning 10 minims at bed time
7th day	12 minims of this solution (bottle No. 3) in the morning 14 minims at bed time
8th day	15 minims in the morning (bottle No. 3) 16 minims (i.e.) at bed time

These injections can be given twice daily as above, or once a day or less frequently. Obviously, however, it is an advantage to get the course of injections finished in as short a period as possible. Because of the possibility of reactions it is desirable that the child should be under observation for a short time after the injection which is best given in bed.

After finishing the course as set out above the child should have the following:—

One week later—1 c.c. of the continuation course solution (bottle No. 3)

Two weeks after this—1 c.c. of the same continuation course solution.

One month after this—1 c.c. of the continuation course solution and each month thereafter 1 c.c. (i.e. 16 minims) for 12 months or longer (bottle No. 3).

In resistant cases it may be necessary to a just the monthly dose and give as much as 24 minims per month i.e., 1½ c.c. (often best given as half the total amount into one arm and the other half into the other arm). At any sign of a general reaction such as wheezing or urticarial rash the next dose should be slightly decreased.

**Prognosis.**—The great majority of cases of asthma in childhood cease before puberty, especially in boys. There are likely to be permanent emphysematous changes with deformity of the chest in some cases.

## HAY FEVER

Hay fever is the reaction of an individual to grass, plant and tree pollen, dust, and other irritating inhalants. Such individuals are sensitive, and are said to be allergic. As a rule, the tendency is inherited. It occurs from the age of one year, and affects both sexes equally.

**Symptoms.**—About the middle of May the sufferers experience an itching or burning sensation in the inner canthus of the eye, or in the naso-pharynx. Violent sneezing with a profuse watery discharge occurs, the eyes become congested, there is a tendency to cough and there may be slight deafness. These symptoms are worse in the morning. In some cases there is, in addition, a tendency to asthma.

**Diagnosis.**—This is made, as in asthma (see p. 162), by skin tests.

**Treatment.**—As in asthma specific desensitization should be carried out. C. L. Bancard Ltd., produce an excellent desensitizing solution for hay fever, as well as suitable testing solutions for the various pollens. Injections should be continued until large doses are being administered hypodermically. This treatment is best given in the first quarter of the year—January to April. In addition, an acid tonic (p. 162) and nasal

<sup>1</sup>G. W. Bray "Hay fever and its treatment in the British Isles," *Med. Press and Circ.* March 29 1922 clxxxv, 1.

ointment are recommended. The same schedule of injections as mentioned on p 162 for asthma, may be used. Failure of treatment appears to be due to insufficient dosage. At any sign of anaphylaxis the dose of the desensitizing solution should be reduced.

**FRIBERINE OINTMENT (U.S. FRED CO.)**

Menthol 5 grains

Eucalyptol 4 minims

Adrenal solution B.P., 15 minims

Ephed. hydrochloride, 4½ minims

Soft paraffin to 2 ounces

A little to be applied to the nostrils night and morning. (Dilute with white vaseline at first if found to make the nose smart.)

For shrinking drops, see p 136

**PULMONARY TUBERCULOSIS**

For tuberculosis of the respiratory system, see Chapter XVI p 331



## CHAPTER IX

### DISEASES OF THE CIRCULATORY SYSTEM

#### HEART DISEASE

**Normal heart-rate.**—A good average for the rate of the heart in childhood is given by Holt as follows

	<i>Pulse rate</i>		<i>Respiration rate</i>
At birth	140-120	At birth	50-32
6 to 12 months	115-100	First year	35-25
2 to 6 years	105-90	2 to 4 years	25
11 to 14 years	85-70	5 to 14 years	25-20

The rate of the heart's action varies with the age and temperament. It also varies with the time, being much faster during the day time than when the child is asleep. To get a true picture of the rapidity of the heart the rate should be taken after the child goes to sleep at night and before he wakes in the morning as it is inclined to be 20 or 30 beats above the normal, even when resting if he is awake. In disease however, this variation between the waking and sleeping pulse rate is much less. For instance, in pneumonia and other acute infections there is no difference at all. The wider the variation between the night and day pulse rate the less likelihood there is of organic disease of the heart.<sup>1</sup>

#### DISORDERS OF RHYTHM

##### Tachycardia (increased heart rate)

(a) **Nervous tachycardia**—Fear, apprehension and excitement will raise the pulse rate of some children over long periods. The diagnosis can be made, however, by taking the sleeping pulse rate, which would be normal.

(b) **Tachycardia due to organic disease**—Infections of all kinds tend to raise the pulse rate temporarily, the variation depending on the age of the child and the height of the temperature. For example, in pneumonia and severe tonsillitis where the temperature is raised to 102° or 103°, the pulse rate may be correspondingly raised to 120 or even 140, and there is no fall during the sleeping hours. On the other hand, in an infection such as rheumatism there may be no fever but the pulse rate is much increased, owing to the action of the rheumatic infection on the myocardium and its nervous control. In diphtheria tachycardia may accompany the acute phase or be found later, accompanying myocardial damage<sup>2</sup> (see p 321). A rapid pulse is characteristic of pink disease and, in fact, may be the most significant symptom in the early stage (see p 242). In military tuberculosis

<sup>1</sup> Cf. A. Sutcliffe and John McMichael. The Pulse-rate and Range in Health and Disease during Childhood. *Quart Jour Med* No. 37 1922 xiv 519

<sup>2</sup> Cf. Neubauer. Bacterial Heart Diseases in Children. *Brit Med Jour*, July 25th, 1914 ii 91

tachycardia is common, both with and without a raised temperature (see p 339). In thyrotoxicosis (Graves's disease) tachycardia is also found (see p 373).

**Paroxysmal tachycardia** may be due to a congenital heart lesion involving the sino auricular node, or may be secondary to rheumatism or other infections of the myocardium. It is rare, the child is brought for intermittent attacks of cyanosis and dyspnoea. In 9 cases described by Hubbard<sup>1</sup> in infants 6 were observed during the first year of life, the heart rate is usually 250 to 300 and if the tachycardia continues for several days (as it is likely to do) it brings on circulatory failure, which is associated with vomiting, dyspnoea, fever, leucocytosis, cardiac enlargement, pulmonary congestion and engorgement of the liver. It may be wrongly diagnosed as pneumonia, or congenital idiopathic hypertrophy of the heart. It may terminate fatally, but responds satisfactorily to adequate doses of digitalis.

**Sinus arrhythmia.**—It is normal to find some degree of sinus arrhythmia in many children, but it is most marked in the thin nervous child. The heart sounds and impulses quicken during inspiration and slow down during expiration. The pause between beats occurs during diastole. The explanation of this type of irregularity lies in the instability of the vagal centre. The diagnosis can be made by the disappearance of the irregularity during rapid breathing or on holding the breath.

**Extrasystoles.**—These may be found in childhood accompanying or following myocardial disease, but more commonly the child appears to be perfectly healthy. On the whole, this irregularity is not of great significance. The regular rhythm is interrupted by the premature beat, which is followed by a longer interval than usual before the next beat. Where rheumatism or diphtheria have affected the myocardium, extrasystoles may be present and should not be looked upon too seriously.

**Bradycardia** (slow heart action).—This is a rare condition which is occasionally familial and on the whole does not indicate heart disease. It may be found in jaundiced conditions and after diphtheria and influenza.

## DISORDERS OF THE MYOCARDIUM

(a) **Cardiac dilatation.**—Normally in childhood the apex beat lies in the fifth inter space in the nipple line, but with cardiac dilatation, which is chiefly ventricular, the apex beat is displaced outward and downward. An X ray will confirm the clinical findings.

**Ætiology.**—Some degree of cardiac dilatation accompanies all acute infections, but the myocardium may be gravely affected in rheumatism, diphtheria, scarlet fever and influenza. In a severe anæmia cardiac dilatation is common, and it may also be found in congenital heart disease, valvular disease of the heart, hyperthyroidism, nephritis, and after violent exercise in a debilitated individual.

**Symptoms.**—With slight dilatation, there may be no symptoms apart from a shortness of breath and tiredness on exertion. Where the dilatation

<sup>1</sup> J. T. Hubbard, "Paroxysmal tachycardia and its treatment in young infants," *Am. Jour. Ped.* (Child) April 1911, 144, 69.

is more marked, there is enlargement of the liver with pulmonary and later generalized oedema and a systolic bruit

**Treatment**—Rest in bed with attention to the underlying cause together with digitalis, will relieve the myocardial strain in most cases

At first, it is necessary to digitalize (saturate) the child and the dose is as follows—0.15 gm of the dried leaf i.e., 22½ minims of Tinct. digitalis per 10 lb body weight, spread over 2–3 days. Maintenance doses are then given on 6 days of each week, each day's doses being 1/10th to 1/15th of the amount used to digitalize the child

*Example*—

Weight of Child	Amount required to digitalize (spread over 2–3 days)	Maintenance daily dose (given on 6 days of the week)
80 lb	$8 \times 0.15 \text{ gm} = 1.2 \text{ gm}$	0.12 to 0.08 grammes
	i.e. $8 \times 22\frac{1}{2} \text{ minims} = 180 \text{ minims}$	38 to 12 minims

If vomiting occurs the maintenance dose should be discontinued for a day or two

(b) **Cardiac hypertrophy**.—Dilatation of the heart suggests weakness of the myocardium, while hypertrophy is a sign of strength and is only found where the more or less healthy myocardium is struggling against some difficulty such as a defective valve. It is also commonly found in adherent pericardium or with congenital heart disease or sclerotic kidneys. The chief guide to the diagnosis is the heaving impulse felt both at the apex and to the right of the sternum and also the X-ray appearances (See Aortic regurgitation p. 168)

## DISORDERS OF THE ENDOCARDIUM

(Cardiac Murmurs and their Significance)

**Cardio-respiratory murmur**.—This is one of the most common murmurs heard in childhood and it is probably formed outside the heart (exocardial). It is thought to be produced by blood forced into the abnormally distended and collapsed pulmonary arteries at the root of the lungs, since the murmur is present on expiration and disappears on full inspiration. It is heard over most of the heart area. It is of no significance and disappears later in life.

**Transient systolic murmurs** may be due to slight cardiac dilatation and accompanied by anaemia. These are often termed haemic and are best heard over the base of the heart.

**Other transient murmurs**.—These may appear during a febrile illness when the heart is functioning vigorously and disappear when the temperature subsides. They are probably due to some slight congenital malformation and play no part in the child's normal life. It is important not to attribute them to an infection of the heart valves, as otherwise a life of invalidism may be needlessly ordered. Such murmurs are often heard just inside the nipple line and are not propagated.

**Murmurs due to inflammation of the heart valves**.—These are most commonly found in childhood associated with acute rheumatism. With recent inflammation the murmur is a soft, blowing one whereas when recovery is taking place, the murmur is louder, harsher or may be musical and widely propagated. The valves damaged are the mitral, aortic, tricuspid and pulmonary in that order of frequency.

(a) *Mitral valve*—Inflammation of this valve gives rise to mitral regurgitation. Where the inflammation is due to rheumatism the valve is seldom involved before the age of 5 years. The murmur is systolic, is best heard over the mitral area and apex, and is propagated into the axilla.

(b) *Mitral stenosis*—Following on mitral regurgitation there is a short or long period when the mitral valve tends to become fibrosed, resulting in a narrowing or stenosis of the opening. This gives rise to the typical crescendo murmur which is pre-systolic, causing a precordial thrill, and in childhood is accompanied by mitral regurgitation with an early systolic bruit. With mitral stenosis and regurgitation there is a loud pre-systolic and systolic bruit which replaces the first heart sound and is followed by a short snapping second sound. Should the cardiac compensation become poor the pre-systolic murmur moves back into diastole.



Fig. 27—Clubbing of fingers in congenital heart disease

and there is now a double murmur (a bellows murmur like the opening and shutting of a bellows) this suggests active carditis.

(c) *Aortic valve*—(i) *Aortic regurgitation* in children this is usually rheumatic in origin and most commonly develops at the age of 9 or 10 years. It accompanies signs of previous mitral disease. It is best heard close to the sternum on the left side. It is usually of grave significance, and is followed first by dilatation and then by hypertrophy of the left ventricle. This produces a very large heart. If the quantity of blood regurgitating through the aorta is considerable, a collapsing pulse will be found. Often the murmur is heard in the mitral area and is thought to be mitral.

(ii) *Aortic stenosis* is said to occur but is extremely rare. The same may be said of

(d) *Tricuspid and pulmonary valve involvement*

## DISORDERS OF THE PERICARDIUM

**Pericarditis** is most commonly caused by a rheumatic infection and accompanies a myo and endocarditis. It may, however, be due to the pneumococcus and be secondary to a pneumonia or to the staphylococcus and follow an osteomyelitis, or it may occasionally follow scarlet fever and tuberculosis.

**Pathology**—Both the parietal and visceral layers of the pericardium are involved. The inflammation causes them to thicken and roughen so that friction results, or there may be an effusion into the pericardial sac. Finally, the two layers of the pericardium may become adherent, or the whole process clear up leaving small pale areas known as milk spots.

(a) **Rheumatic pericarditis** *Pathology*—Both layers of the pericardium (visceral and parietal) become thickened and inflamed, with a tendency to be shaggy and adherent. Small loculated collections of fluid form in the pericardial sac. Along with the inflammation of the pericardium a rheumatic mediastinitis occurs with glandular enlargement locally. With restricted movement of the heart, there is a tendency to dilatation and hypertrophy. Recovery leaving a smooth glistening pericardium is possible, but improbable. (For clinical picture, see p 361.)

(b) **Pericarditis due to pyogenic organisms** (pneumococcus and staphylococcus) *Pathology*—In such cases there is much thickening of the pericardial layers and a considerable effusion of pus occurs at once. The pericardial sac becomes greatly distended and the heart sounds are much muffled. The pus may form large clots and the so called "bread and butter" appearance of the visceral and parietal pericardium is characteristic. Where successfully treated, the pericardium appears to be able to recover completely.

**Clinical picture**—It is characteristic that pericarditis is quite unsuspected. The child is found to be much more ill than either the pneumonia or osteomyelitis would suggest. An examination of the heart in the early stages reveals a rough to and fro friction sound and, later, muffled heart sounds. An X ray shows a much enlarged heart shadow. The temperature is high, the respirations and pulse-rate rapid and the child shows signs of marked toxæmia.

**Medical treatment**—If given early and in adequate doses, the Sulpha drugs claim a number of successes (for dose, see p 383).

**Surgical treatment**—It may be necessary to drain the pus from the pericardium and this is often a very satisfactory method of dealing with such cases. It is usually done by open operation rather than by aspiration.

CONGENITAL HEART LESIONS<sup>1</sup>

## (Congenital Morbus Cordis)

Congenital heart malformations are due to some intra uterine developmental defect, the cause of which is at present obscure. The diagnosis, except in a few clear-cut cases, is extremely difficult, in fact, in the vast majority of cases one can but hazard a guess of the nature of the lesion, and the post mortem examination often shows this to be entirely wrong.

<sup>1</sup> G. A. Sutherland and J. A. Osler, Batten, Thomsell and Peterson's "Diseases of Children, 3rd Ed., Arnold & Co., p. 353.

The right side of the heart is much more commonly affected than the left side

#### LESIONS CHARACTERISED BY CYANOSIS

**Pulmonary stenosis**—This is the second most common of the congenital malformations and gives rise to a typical clinical picture. At birth or shortly after the infant is seen to be cyanosed and clubbing of the fingers and toes rapidly develops, and later even clubbing of the nose and lobes of the ears. The respirations are rapid, the lips and tongue are dark blue and the gums are extremely congested. The liver is enlarged. The præcordial dullness on percussion extends to the right of the sternum showing the enlargement to be mainly that of the right side of the heart. There is a tendency to epistaxis or sudden hæmorrhage from the mucous membranes. Slight pulmonary infections often prove rapidly fatal. A loud harsh systolic bruit is heard over the basal portion of the heart in the region of the pulmonary artery, and is propagated down the sternum.

**Pathology**—The pulmonary artery carrying venous blood to the lungs is stenosed to a greater or lesser extent, thus there is deficient aeration of the whole of the blood stream giving rise to the cyanosis. In an attempt to combat the stenosis the right side of the heart hypertrophies.

**Tetralogy of Fallot**—This is found in older children and young adults and consists in pulmonary stenosis, hypertrophy of the right ventricle, a patent interventricular septum and an aorta which opens into the right ventricle as well as the left. The blood passes partly from the right ventricle through the stenosed pulmonary artery to the lungs, but also from the right ventricle to the systemic system through the aorta. A basal systolic murmur is present and the patient may survive even to middle life.

#### POTENTIAL CYANOTICS

**1 Patent interventricular septum (Maladie de Roger).**—There is a harsh systolic bruit and thrill over the heart. The blood tends to pass from left to right. Consequently there is no clubbing or cyanosis. The heart may be very slightly enlarged and the activities of the child are unimpaired.

**2 Patent ductus arteriosus**—The murmur heard over the base of the heart is characteristic, and a loud humming bruit extends through systole and diastole. The heart may be slightly enlarged, the blood flows down the patent ductus arteriosus which passes from the arch of the aorta to the left branch of the pulmonary artery. The blood is therefore flowing from the systemic to the pulmonary circulation and consequently there is no cyanosis or clubbing. There may be few if any symptoms of heart disease. Because of the fear of sub-acute bacterial endocarditis the operation of tying off the ductus arteriosus has been performed with some measure of success in recent years.<sup>1</sup>

**3 Patent interauricular septum.**—Very commonly, there is a patent foramen ovale without symptoms, since the opening is almost sealed off by its valve or membrane. Patent interauricular septum, however, may

<sup>1</sup> R. E. Gross, "Patent ductus arteriosus and its surgical treatment," *Advances in Paediatrics*, Helmsmann, 1947, Vol. 1, p. 14.

constitute a slight or gross defect, and give rise to definite symptoms, as the blood flows from the systemic to the pulmonary circulation, i.e. from the left to the right side of the heart, distending the right auricle and right ventricle, which in turn become hypertrophied, along with the pulmonary artery. There is a well marked systolic murmur. Holt and McIntosh state that such individuals are susceptible to rheumatic fever, cardiac arrhythmias and pulmonary infections, and an X ray shows increased hilar shadows.

#### LESIONS CHARACTERIZED BY ACYANOSIS

1 **Coarctation of the aorta**.—This is rare, the aorta is narrowed or completely stenosed, with a well marked collateral circulation which gives rise to a variety of symptoms and different clinical pictures according to the level of the stenosis. Usually a murmur is to be heard between the scapulae, and the pulse at the wrist is good while that in the inguinal regions or popliteal space is poor or absent. An X ray will show notching or grooving of the ribs, due to hypertrophy of the intercostal arteries which may be greatly enlarged. The prognosis for life in cases surviving the first year seems to be good.

2 **Dextrocardia**.—This very rare condition may occur by itself or be accompanied by transposition of the viscera. In the latter case the liver and spleen are reversed in position and the stomach lies on the right. The heart lies to the right of the sternum and gives a characteristic electrocardiogram. There may be no symptoms and the condition may be discovered by chance.

**Diagnosis of congenital heart disease**.—*Congenital murmurs* are usually *systolic* and *best heard over the base* and are much more pronounced and harsh than those of acquired lesions. On the whole it is true to say that murmurs discovered by chance play no part in a child's life. Often a child, showing no disturbance of his general health, is discovered to have a loud basal systolic murmur. An acquired lesion, severe enough to produce such a murmur, would have caused very serious disturbance of the general health and could not possibly have escaped detection. On the whole *acquired lesions* are *apical*, and rarely occur before the age of 5 years.

*Cyanosis* of any marked degree is accompanied by a polycythæmia, the red cells being increased to from 6 to 9 million per c mm. As a guide to the severity of the lesion, the degree of cyanosis, the size of the heart and the rapidity of the respirations after exercise must be noted.

*Infantidism* may result from severe congenital morbus cordis. Fifteen per cent. of all Mongols have congenital heart defects. Murmurs may be heard during the neonatal period, and are probably due to a patent ductus arteriosus. Such murmurs entirely disappear with the closure of that vessel, at the third or fourth week.

**Electrocardiogram**.—In doubtful cases an electrocardiogram will often differentiate between a congenital and an acquired lesion.

In the infant screaming attacks are common. These may be compared to the anginal attacks of the adult. When such an attack occurs the

\* W. Evans, "Congenital Stenosis (Coarctation), Atresia and Interruption of the Aortic Arch." *Quart Jour Med.*, N. 3 Jan 1933 B 22.

screaming continues for some minutes and then suddenly subsides. This may be accompanied by a greater or lesser degree of constipation, which should be avoided.

**Prognosis.**—The infant with a severe congenital heart lesion starts life very much handicapped. Its road is beset with difficulties, in infectious diseases, and it may have to surmount a pulmonary infection. Such infections throw added strain on an already damaged and overworked organ, and a large proportion of infants with the severer lesions succumb in the first few weeks or months of life. Those showing rapid respiration as infants, before the crawling or walking stage, are unlikely to survive any length of time. Those who appear perfectly healthy, but show slight dyspnoea or cyanosis on walking or running, may survive for a considerable time, but a careful examination will show that their compensation is poor, the heart enlarged, the liver congested and increased in size, and the circulation imperfect. The outlook is good where the lesion is discovered by accident in a routine examination or where shortness of breath has been discovered on violent exertion in early or late childhood.

A certain proportion of cases of congenital heart disease later develop malignant or subacute bacterial endocarditis. The remote prognosis is affected by this tendency.

**Treatment. Posture**—The position in which the infant is placed to rest in its cot is important. Some are comfortable and will sleep well on one side or the other, but there are a certain number who rest only lying across a pillow, with the head hyperextended.

**Gain in weight**—The gain is extremely small in the majority of cases as, owing to weakness and dyspnoea, the infants are unable to take sufficient at a feed. If breast fed, they tend to underfeed themselves, and the breast milk to fail. It is well to feed at short intervals, and 7 or even 8 feeds may be necessary in the 24 hours. Concentrated feeds of high caloric value are an obvious advantage. After having failed to gain for some months, or having gained only very slowly, there may be, at about a year, a marked improvement, many ounces being put on each week. Certainly the more solid the food, that is, the more concentrated, the better the progress.

**Exercise**—Each individual case must be studied before directions for exercise can be given. As a rule, the child will itself tend to restrict its energies, owing to its obvious discomfort. The use of a push chair when out for an airing should be continued in these cases long past the ordinary age.

*In the majority of cases of congenital heart disease no specific treatment is required throughout life.*



## CHAPTER X

### BLOOD DISEASES<sup>1</sup>

**Normal blood picture in infancy<sup>2</sup>**—Blood is formed during intra uterine life not only in the bone marrow, but also (extramedullary) in such organs as the liver and spleen and sometimes the kidneys along with other portions of the reticulo endothelial system. The whole of these blood forming sites including the blood itself with all its cells has been designated 'the erythron'.

During foetal life the oxygen tension in the foetal circulation is low and the infants react in the same manner as individuals who reside in high altitudes namely by an increase of red cells up to 6 or 7 millions. After the infant is born both the red cell and the haemoglobin content of the blood fall rapidly as they are not required since the oxygen tension has been raised and at the same time the circulating nucleated red cells present before birth rapidly disappear from the blood stream.

Occasionally, jaundice (icterus neonatorum) develops because of the presence of bilirubin in the blood plasma as a result of this physiological destruction of blood cells. This soon passes off.

#### ORIGIN OF THE RED CELLS (NORMAL ERYTHROPOIESIS)

The red cells develop from an intravascular endothelial cell in the reticulum of the bone marrow. This endothelial cell develops into a megaloblast then an erythroblast, a normoblast, a reticulocyte and an erythrocyte. While the mature cell is developing it diminishes in size and acquires haemoglobin.

#### ORIGIN OF THE LEUCOCYTES (NORMAL LEUCOPOIESIS)

The granular series of leucocytes develops extravascularly from primitive white cells, myeloblast myelocyte and finally to the neutrophil eosinophil, basophil polymorphonuclear.

The white cells are amoeboid and enter the circulation through the walls of the intersinusoidal capillaries.

Lymphocytes (according to Whitby) develop in germ centres in lymphatic glands through the stages of primitive white cell lymphoblast large lymphocyte small lymphocyte and enter the circulation by the thoracic duct.

Monocytes (often called large mononuclears or transitional cells) arise from a reticulum cell mainly in the spleen and to a lesser extent in the bone-marrow. The primitive cell the monoblast gives rise to a monocyte.

Blond platelets are developed by budding from megakaryocytes, which are formed by a fusion of several reticulo endothelial cells which have probably an extravascular origin.

**Notes**—(1) The development of monoblast and monocyte on the above lines is by no means universally accepted.

(2) The term megaloblast is restricted by some haematologists to an embryonic cell or a pathological cell.

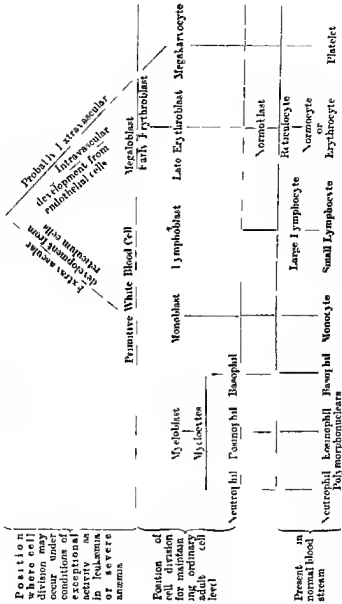
**Haemoglobin**—Helen MacLay has found (see Fig. 30 p. 177) that at birth the haemoglobin is about 140 per cent. (Haldane). By the age of 2½ to 3 months it has

<sup>1</sup> F. J. Poynton, H. L. Thurnfield and Donald Paterson. The Severe Blood Diseases of Childhood. *Brit. Jour. Child Dis.* 1934, six 3, 1-8 and 178. Garrair, Estlin, Thurnfield and Paterson's "Diseases of Children" 3rd Edn., Arnold & Co. p. 462.

<sup>2</sup> L. G. Larnock and W. O. Smallwood. "Anaemia in Infancy and Childhood." *Practitioner* March 1935, cxliii, 398.

ORIGIN OF BLOOD CELLS \*

Reticulo Endothelial System in Bone Marrow, Spleen Lymph Glands etc



\* From " Disorders of the Blood, by L. F. H. Whitty and C. J. C. Britton (Clarendon), 3rd Ed

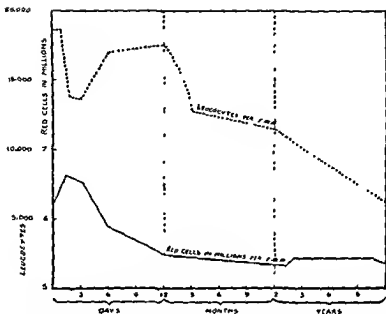


Fig. 28.—Red cell and leucocyte counts from birth to 12 years of age  
 Reproduced by permission from "Disorders of the Blood" by L. E. H. Whitby and C. J. C. Britton  
 (Churchill), 3rd Ed.

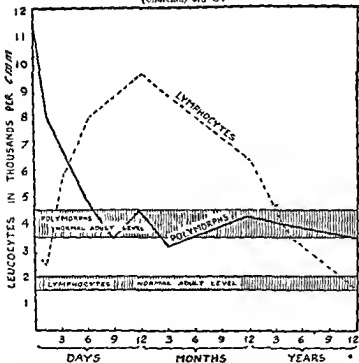


Fig. 29.—Absolute numbers of polymorphonuclear and lymphocytes per c.mm.  
 from birth to 12 years of age.  
 Reproduced from "Disorders of the Blood" by L. E. H. Whitby and C. J. C. Britton  
 (Churchill), 3rd Ed.

dropped to nearly 70 per cent, but from then on rises so that by the sixth month it is stabilized at approximately 85 to 90 per cent.

**Red cells** (see Fig. 28).—Along with the fall in hæmoglobin there is a fall in the red cell count from approximately 7 million to 5½ million by the twelfth day. From that time on, the red cells remain more or less stationary up to the age of 10 years.

**Leucocytes** (see Fig. 28).—During the first 12 days of life the total leucocyte count is approximately 18 thousand, but from this time on there is a steady fall, so that by the twelfth year the total leucocyte count is similar to that of an adult, i.e. 6 thousand per cmm. The proportions of polymorphonuclear and lymphocyte cells, however, which at birth is that of the adult, rapidly reverses itself by the twelfth day. It is not until the sixth to ninth year that the adult high polymorphonuclear and low lymphocyte count is regained.

**Bone marrow**.—At birth all the bone marrow is of the red hæmopoietic type, and it is packed with nucleated red cells. By the twelfth year the distal bones have begun to assume the adult type of yellow (fatty) marrow, and this is complete by the twentieth year. Red marrow remains in the adult only in the bones of the skull and thorax. In the infant the bone marrow is working actively, therefore, at all times. If there is a fresh call for extra hæmopoiesis, extramedullary centres must answer it. Such centres as the liver, spleen and kidneys must consequently resume their hæmopoietic activity, and as a result many primitive cells are thrown out.

**Blood-platelets**.—These are present at birth and number 250,000 to 500,000, as in adults.

**Coagulation time**.—This is prolonged for the first ten days of life.

**Bleeding time**.—This is normal as in adults.

## ANÆMIA

Anæmia is the failure of the blood-forming tissue to maintain the normal supply of blood corpuscles and of hæmoglobin to the rest of the body, and its severity is measured by the failure of the capillary blood to reach certain normal standards in respect of numbers of corpuscles and amount of hæmoglobin.

**Clinical picture and diagnosis**.—This is sometimes difficult, as differentiation must be made between pallor of the skin and anæmia. Children with sunburnt hair or very fair children often give a false impression, and only when the hæmoglobin falls to well below 50 per cent, sometimes are such symptoms noted as shortness of breath on exercise, together with enlargement of the liver. The examination of the palms of the hands is of particular help, also of the gums and conjunctiva. The student should examine the sclerotics for jaundice and palpate the abdomen carefully for enlargement of the spleen. A hæmic murmur is usually present over the heart, especially in the basal region, if there is any anæmia. Often there is a slight fever. Some œdema of the lower limbs develops if the anæmic child is allowed to run about. There is a tendency to complain of undue tiredness.

## CLASSIFICATION OF ANÆMIAS (PARSONS)

**A Hypoplastic an hæmatopoietic or deficiency anæmias**.—This group is characterized by defective blood formation, which may be due to a deficiency of iron, vitamins or internal secretions.

**B Hemolytic or erythronoclastic anæmia**.—This is characterized by hæmolysis and destruction of the blood cells in the blood stream or actually in the bone marrow.

<sup>1</sup> G. R. Minot and W. B. Castle. "Interpretation of the Erythrocytic Reaction." *Lancet* Aug. 10, 1933.  
Joseph, "Mechanism of Anæmia in Infancy." *Bull. Johns Hopkins Hosp.*, No. 4, Oct. 1937, p. 185.

## DEFICIENCY OR ANHYMATOPOIETIC ANÆMIAS

1 **Nutritional anæmia** (*Hypochromic microcytic anæmia of infancy* milk anæmia) —This is the commonest anæmia of infancy

**Ætiology** —It is due to a diet deficient in minerals, particularly iron and occasionally copper. Infants who are too exclusively milk fed (either breast or artificial milk) develop this anæmia. It is also seen in premature infants, and twins who have been born before a proper store of iron has been given to them by their mother.

It is often seen in the very badly managed infants or mental defectives who have refused solid food and been fed on milk only. Occasionally it is seen in older children whose diet is deficient. It is suggested by Lightwood<sup>1</sup> that such children may not absorb their iron because of a hypochlorhydria or a coincident chronic infection.

**Clinical picture** —Such infants are extremely pale although quite plump and well grown. If the condition has continued for any length of

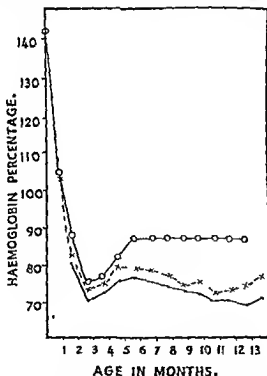


Fig. 30.—Haemoglobin curves

The graph shows the fall in haemoglobin during the first three months of life—the physiological anæmia of infancy—and the development of nutritional anæmia after the age of 6 months. The effect of iron treatment in the prevention of nutritional anæmia is demonstrated. (*Helena Vucelja*)

x — x = average haemoglobin curve breast fed  
 — — — = haemoglobin curve artificially fed.  
 o — o = haemoglobin curve iron group

<sup>1</sup> R. Lightwood and J. C. Hawkesley *Arch. Dis. Child.*, 1934 ix 239

time the spleen is palpable and there is a loud hæmic murmur over the heart. This may be so marked that heart disease may be suspected. Such infants suffer from colds and infections and Helen Mackay<sup>1</sup> claims that these are due to the anæmia.

From the work of Mackay it appears that the average infant whether breast or bottle fed tends to develop some degree of nutritional anæmia unless given iron. Fig. 30 shows the good effect of iron therapy on the hæmoglobin content of the normal infant's blood. In addition to the murmur and pallor such children are short of breath on exercise and they are always tired.

**Blood examination**—The red count is normal or only slightly reduced. It is the hæmoglobin which is grossly deficient. The cells are therefore small and contain very little hæmoglobin. The colour index varies between 0.7 and 0.8, the hæmoglobin being perhaps from 50 to 20 per cent. Stained films show the cells to be hypochromic and microcytic. Poikilocytosis is usually present.

**Treatment** *Feeding*—Mixed feeding, with cereals e.g. green vegetables particularly spinach and broth should be instituted at the earliest possible moment. The inclusion of iron in some of the dried milks as in *Hæmolac* and *Ferrolac* adds a grain or more of iron and ammonium citrate to the child's diet in the 24 hours.

*Iron*—Smallwood<sup>2</sup> suggests 1 grain of ferrum redactum mixed with fine sugar placed on the back of the tongue from one to three times daily. For older children from 2 to 10 years the following prescriptions are useful.

R Ferri cati sacch

As much as will lie on a shapence t.i.s.p.c.

This should alternate with

P Ferroch. (A & H)

One teaspoonful t.d.s.p.c.

Or

R Reduced iron ʒi grains

Hydrochloric acid (B.P.) 110 minims

Water to 1 fluid oz.

Liqui glucose 1.0 minims

Syrup (B.P.) to 5 or 6 drachms

60 minims t.d.s.p.c.

This might alternate with

R Iron and ammon. cit. 5 grains

Syrup 30 minims

Water to 60 minims

60 minims at d.s.p.c.

These would both be best given diluted with water and sucked through a straw.

*Copper*—It has been shown that certain metals having approximately the same atomic weight as iron e.g. copper and manganese, act as catalysts and cause rapid and very efficient absorption of iron. The following mixture is satisfactory.

P Copper sulphate ʒi gr

Mang. chloride ʒss gr

Comp. syr. of phosphate of iron ʒo minims

30 minims at d.s.p.c.

<sup>1</sup> Helen M. Mackay "Nutritional Anæmia in Infancy" with special reference to Iron Deficiency. Med. Research Council 1933. H.M. Stat. Office.

<sup>2</sup> G. Smallwood "Practitioner" April 1933 vol. 43a.

This preparation is highly recommended for older children

R. Hæmatomic Plastiles (John Wyeth)

One to be given t. d. s. p. c. crushed up in jam or honey

Newborn infants should be given the following before they reach the age of one month

R. Halibut liver oil

Two drops t. d. s. a. c.

to alternate with

P. Iron and ammon. c. t. 5 grains

Syrup 30 minims

Water to 60 minims

Three to 10 drops t. d. s. a. c. or one teaspoonful t. d. s. p. c.

or

R. Ferrous sulphate 1½ gr.

Acid hypophosphorous d. l. ½ minim

Syrup 50 minims

Aq. aurantis ad 60 minims

1 day of each to be given in rotation

It is well to put older children with anæmia to bed as dilatation of the right side of the heart and congestion of the liver are usually present. Much more rapid progress is made when this rest is insisted upon. Sunbath, fresh air and a well balanced diet are essential.

**2 Anæmia of celiac disease**—A proportion of cases of celiac disease show a megalocytic hyperchromic type with a palpable spleen. These cases do best on liver such as Campolon 4 ccm intramuscularly or Proethron forte 2 c.c. per week and Marmite or other vitamin B complex preparation such as B plex (John Wyeth) daily.

The other type of anæmia is an iron deficiency one such as has just been described above. Such cases do extremely well on massive doses of iron.

**3 The anæmia of cretinism and scurvy**—This is an orthochromic type, in which there is a coincident fall in the hæmoglobin and red cell count leaving the colour index at approximately 1.0. In such cases the manufacture of the red cells appears to be slowed down. The missing factor in cretinism is thyroxine and in scurvy it is vitamin C.

**4 Anæmia after severe loss of blood**—When there has been bleeding after tonsillectomy, circumcision or dental extraction or with such diseases as purpura or hæmophilia a hypochromic microcytic anæmia develops, as described under 1.

**5 Pernicious anæmia** is not definitely known in infancy. In those cases claimed there is probably some other cause (Smallwood and Whitby).

#### HÆMOLYTIC OR PYRROCYTIC ANÆMIA

In hæmolytic anæmias the chief characteristics are

- 1 Increased bilirubinæmia
- 2 Some degree of jaundice
- 3 A positive indirect van den Bergh reaction
- 4 Urobilinogen in the urine
- 5 Anæmia of orthochromic type with a reticulocytosis as a rule
- 6 Splenomegaly
- 7 The destruction of the red cells in the circulating blood with some damage to the bone-marrow

1 **Erythroblastosis** (*Icterus gravis neonatorum*, *Hydrops foetalis* and *anæmia hæmolytica*)—(See p. 68, "Anæmias of the Newborn")

2 **Von Jaksch's anæmia** (*Splenic anæmia of infants* *Anæmia pseudoleukæmia infantum*)—This is probably not a separate entity, but a subacute or chronic acquired hæmolytic anæmia of infancy and early childhood (Smallwood). The characteristics are an abundance of nucleated red cells, and immature white cells of the myeloid series in the blood stream. There is also splenic enlargement, and occasionally purpura. It is found in premature infants, twins, and associated with rickets and chronic infections. The child is usually between one and three years of age, and has a large abdomen with an enlarged liver.

**Treatment**—A good diet, containing an abundant supply of vitamins, together with fresh air, sunshine and iron, are indicated. Transfusions of blood at intervals will accelerate the cure. The transfusion is best given by the drip method, and 10 c cm per pound of body weight should be sufficient.

These cases ultimately recover completely. They are subject however, to intercurrent infections, which may carry them off.

3 **Acute hæmolytic anæmia: type Lederer**.—This is a very rare condition, most commonly seen between the age of 6 months and 3 years. The onset is sudden, and there is fever, jaundice, and sometimes blood in the urine. The blood shows a leucocytosis, with some myelocytes. There is a marked reduction of the red cells to one or two million. The condition is probably infective.

**Treatment**—Transfusions by the drip method of 10 to 15 c cm per pound body-weight are indicated.

4 **Erythroblastic anæmia of childhood: type Cooley**.—This is a hæmolytic anæmia with a blood picture like that of von Jaksch's anæmia, but it is almost exclusively found in children of Mediterranean origin. It is extremely rare in this country. The blood shows anæmia, with a remarkable increase in nucleated red cells.

**Clinical picture**—Splenic enlargement, curious pigmentation of the skin throughout, giving a Mongolian appearance, with cranial and nasal bones much thickened due to a hyperplasia of the bone-marrow. There is no adequate treatment. Death occurs about the tenth year.

5 **Sickle-celled anæmia**.—A rare condition occurring almost entirely in negroes. The red cells show a sickle shape. There may be abdominal pain, joint pains, and ulceration of the legs. Exacerbations occur at intervals.

**Diagnosis**—This is made by examining a fresh sealed specimen of blood.

**Treatment**—This is symptomatic. Blood transfusions help during an acute phase. Death takes place in active cases from intercurrent disease. Latent cases may live many years.

6 **Acholic family jaundice**<sup>1</sup> (*Congenital hæmolytic anæmia*).—Whitby and Britton define this as a chronic familial disease characterized by crises of excessive blood destruction, a constant high percentage of circulating reticulocytes, splenomegaly, spherocytosis, fragility of the red

<sup>1</sup>T. W. Lloyd "On the Etiology of Acholic Family Jaundice, Oxford University Thesis, 1911



cells, and a variable amount of jaundice, but with no bilirubin in the urine

**Ætiology and blood changes**—The cause is unknown, but it is supposed that there is a congenital defect in the make-up of the erythron. The red cell envelopes are unduly fragile, and the cells themselves are spheroidal, and not dumb bell shaped as is usual. There is microcytosis. Bilirubin is present in the serum, the icterus index is high and the van den Bergh test is of the indirect type.

The reticulocytosis may be as high as 20 to 50 per cent. After splenectomy there is a clinical but not a biological cure.

**Pathology**—The spleen is much enlarged. The liver, spleen, kidneys and bone marrow contain an excess of iron and they give a strong Prussian Blue reaction. Gall stones are not uncommon (D Gardiner, 1933). When they do occur, they complicate the clinical picture by exaggerating the jaundice and a direct van den Bergh is present.

**Symptoms**—At three or four monthly intervals crises occur in which the child (who may be of any age from infancy up) becomes anæmic and mildly jaundiced. There is weakness and the child is feverish and ill, and at these times blood changes occur and there may be vomiting. It takes three or four weeks to recover.

**Differential diagnosis**—From epidemic hepatitis by van den Bergh, colour of stools, anæmia, reticulocytosis, fragility of red cells, splenic enlargement, and family history. When there are gall stones, the differentiation is not so easy.

**Typical example of acholuric family jaundice**—A P, a boy aged 6 years, had been jaundiced and pale from birth, and jaundiced at intervals since. Both the mother



Fig. 31 Acholuric family jaundice. A P, aged 6 and his sister aged 4 years. The costal margins are marked out and also the spleen.

and grandmother were said to be jaundiced and pale. There are three children in the family, a younger sister aged 4 years and a brother aged 7 months. The sister has a large firm spleen and her fragility is increased; the brother has a palpable spleen but his fragility is normal. The patient has a large firm spleen, and the urine contains urobilin. His fragility is markedly increased. A splenectomy was done. The fragility test although it did not become normal showed marked improvement after operation and the child developed a good colour complete recovery apparently taking place (Fig 31.)

**Treatment**—If the general health is good, nothing need be done beyond giving iron. If, however, the child is a chronic invalid, splenectomy is warranted.

**7 Anæmia of acute and chronic infections**—Accompanying acute streptococcal infections of the throat with septicæmia also pyelitis, and chronic infections such as tuberculosis and rheumatism there is a varying degree of anæmia. This may be due to actual destruction of the red cells or to the action of toxins on the blood forming tissues bone marrow, liver and spleen.

As a rule, an orthochromic anæmia results with a colour index of between 0.7 and 1.0. Iron has little or no effect in such cases. Treatment for removal of the underlying infecting focus is required.

## PURPURA

### ESSENTIAL THROMBOCYTOPENIA

(Purpura hæmorrhagica, Werlhof's disease)

Primary thrombocytopenic purpura hæmorrhagica)

**Definition** (Whitby and Britton)—A disease characterized by multiple hæmorrhages into the skin or from the mucous membranes associated with a reduced platelet count, a prolonged bleeding time, a normal coagulation time, and a lack of proper clot retraction.

**Incidence**—It is most common in children and young adults.

**Ætiology and pathology**—The reduction of the platelets is probably the basic factor. This may be due to a defective production of platelets by the bone marrow, or an excessive destruction by the spleen. Another explanation is that the platelets are themselves defective.

**Clinical picture**—Usually the disease is chronic, and there are frequent mild attacks of bleeding. In the acute form purpuric patches appear over the surface of the body and the mucous membranes. Bleeding takes place from the nose, alimentary tract or urogenital tract (hæmaturia). The child bruises easily from slight knocks. The capillary resistance test is positive. The platelets are reduced below 40 000 per c.m., which is the critical level at which purpura and hæmorrhage takes place. The bleeding time is much prolonged. The clotting time is normal but the clot is a poor, soft one.

The general health is on the whole fairly good although these children tend to be somewhat anæmic.

**Treatment**—The mild chronic cases seem to do well and require nothing more than iron tonics. The severe cases need repeated transfusions and, should there be recurrences it is best to have the spleen removed. When possible the operation should be done in an interval between attacks. X rays to the spleen are said to be beneficial. Vitamin

C, vitamin P snake-venom, liver and calcium all have their advocates. None however, seems to be really effective in a severe case.

**Differential diagnosis**—Other purpuras may be excluded by the fact that pain in the limbs and joints is not a feature. The reduced platelet count and the prolonged bleeding time are characteristic. Aplastic anaemia may be excluded by the fact that in that disease all the blood elements are diminished and there is no sign of regeneration as shown by reticulocytosis.

Sternal puncture will confirm the diagnosis as it is normal in thrombocytopenia but aplastic in aplastic anaemia.

**Hæmophilia**—In this disease the platelet count, capillary resistance test and bleeding time are normal but the coagulation time is prolonged.

**Acute lymphatic leukaemia** especially when there is an accompanying leucopenia may superficially resemble thrombocytopenic purpura. The course of the disease and further blood counts will distinguish the two conditions.

**Agranulocytic angina**—In this condition there is a necrotic condition of the mouth and there is no reduction of the platelets.

#### SYMPTOMATIC THROMBOCYTOPENIA

This condition may be due to

1 **Bone-marrow defect**—Examples of this are seen in aplastic anaemia, leukaemia, benzol and other poisoning, the effect of X rays, radium and the toxins of fever.

2 **Splenic defect**, as seen in Banti's disease and Ganther's disease.

3 **Capillary endothelial damage**—Examples of this occur after the toxæmia of fever (such as small pox), snake poisoning and some drugs (such as belladonna and quinine). The purpura of scurvy is due to a lack of vitamin C which allows the capillary endothelium to become unduly permeable. Suffocation and convulsions cause bleeding because of an undue strain on the capillaries.

4 **Anaphylactoid purpura** (Henoch-Schönlein syndrome)—Under this heading are grouped (a) purpura simplex, (b) Henoch's purpura, (c) Schönlein's disease, (d) allergic purpura. The essential in this group is one or more of the following symptoms: urticaria, oedema of the subcutaneous or submucous tissues, joint pains, or abdominal manifestations.

**Ætiology**—The allergic agent may be a food or infection. It is often difficult to ascertain.

**Symptoms**—There is usually malaise, headache and fever during an attack. In simple purpura spots on the limbs are often the only manifestation. Joint pains may accompany them. In Henoch's purpura there is usually marked abdominal pain and appendicitis may be simulated. There may be melæna, and because of this, intussusception may be suspected. A close examination of the body will show the presence of purpura, and very often the child has a clean tongue. In Schönlein's purpura (purpura rheumatica), acute rheumatism is simulated closely. The joints are swollen and painful. There is a slight fever, and some purpura of the

skin Nephritis (hæmaturia) may be present in any of the above conditions, and this is a serious complication

Treatment—Beyond rest in bed during an acute phase, no treatment appears to be of any value

### HEMORRHAGIC CONDITIONS DUE POSSIBLY TO A QUALITATIVE PLATELET DEFICIENCY

**Hæmophilia**—Definition (Whitby and Britton)—An hereditary disease affecting males but transmitted by females, and characterized by a prolonged coagulation time, and a lifelong tendency to excessive hæmorrhage

**Ætiology**—The tendency to bleed is a Mendelian sex linked recessive character. The male children of mothers who have inherited this characteristic will all be bleeders. The female children of hæmophilic fathers will be able to transmit the tendency to bleed to their male children in turn. Latin races appear to be exempt (Whitby)

**Pathology**—The coagulation of the blood is markedly delayed. The hæmophilic platelets do not break up and liberate thrombokinase as they should. The platelets are normal in number.

**Symptoms**—These usually appear at about two or three years of age. A steady slow bleeding takes place from a trivial cut, or after an operation for circumcision or dental extraction. Epistaxis is common as is hæmorrhage into the joints, the knees and elbows being most affected. The joints are swollen, red, tender and the child is feverish. On the first or second occasion the blood is absorbed but subsequent bleeding tends to produce a partly ankylosed joint. (See p 284) Large hæmorrhages may take place beneath the skin, or into the buttock or abdominal wall. The spleen is not enlarged. The capillary test is negative, and the blood calcium is normal.

**Diagnosis**—This is made by the fact that males only are affected, and there is usually a family history. The blood coagulation time is much prolonged, up to several hours. Thrombocytopenic purpura is excluded by the normal platelet count, and the absence of purpura.

**Prognosis**—There is a general tendency to improve with age. The mortality is high before puberty. Permanent joint damage is the rule.

Treatment—The immediate treatment to be followed is

- (1) Application of a warm cloth, soaked in normal blood, to the bleeding area
- (2) Application of 1 in 10 000 solution of snake venom (B W & Co) to the area
- (3) Intramuscular injection of 10 to 20 c cm of normal blood
- (4) Transfusion with normal blood is best of all

Many prophylactic measures have been advocated, but none seem definitely of value

### AGRAULOCYTIC ANGINA

**Definition.** (Whitby)—A severe disease characterized by marked leucopenia, due to extreme diminution or absence of cells of the myeloid series, and associated with necrotic ulceration, particularly of the mouth. Drugs as a cause should be excluded.



Plate 9 — Rash of purpura



Plate 10.—Pyelogram of a case of pyonephrosis.

The left kidney is greatly enlarged and both the pelvis and the calyces are much dilated.

**Blood picture** —The leucocytes fall to less than one thousand, and the polymorphonuclears are almost or entirely absent. The mouth and throat present a sloughing ulcerated appearance.

**Prognosis** —In children the mortality is high, probably 80 per cent.

**Treatment** —Blood transfusions and large doses of pentnucleatide hypodermically are used as a bone marrow stimulant, to increase the production of the granular series of leucocytes. From 10 to 50 c cm of the solution are given daily. An improvement is said to occur on the third to fifth day.

**Differential diagnosis** —It is difficult to distinguish cases of leukaemia with a marked leucopenia. Bone marrow puncture is indicated.

#### APLASTIC ANÆMIA

**Definition** (Whitby) —A severe condition characterized by extreme anaemia, leucopenia and thrombocytopenia, due to aplasia or hypoplasia of the bone marrow. The disease occurs in an idiopathic or primary form of unknown aetiology, and as a condition secondary to certain bone marrow poisons.

#### Idiopathic Aplastic Anæmia

**Aetiology** —It is suggested that in young people the bone marrow may be congenitally weak and easily exhausted, while in adults it is usually infection, and poisons which damage the bone-marrow.

**Pathology** —The bone marrow loses its red hæmopoietic appearance and is replaced by a grey or yellow 'fatty' marrow. The liver, spleen and the lymph glands are unaltered.

**The blood picture** —There may be a reduction of red cells and hæmoglobin and of white cells at the same time, or the white cells may be normal. Signs of regeneration, in the form of reticulocytes, are almost or entirely absent. The platelets may be reduced. The blood picture is a reflection of the bone marrow condition. Sternal puncture should always be carried out.

**Symptoms** —The symptoms are those of anaemia. There may be hæmorrhage, and purpura, as the condition progresses. The adrenaline test, with 15 minims of 1/1,000 adrenaline, should be made. In a normal person, blood counts done at ten minute intervals after the adrenaline will show a rise both in red and white cells and the presence of young leucocytes in the peripheral blood. This will not be the case in aplastic anaemia.

**Prognosis** —Usually, this is hopeless in the true idiopathic type.

**Treatment** —Transfusions will keep the child going, and as many as fifty or a hundred have been given to children during a year. Whitby recommends splenectomy when the child has been built up with transfusions.

#### Symptomatic Aplastic Anæmia

Symptomatic aplastic anaemia may be brought about by benzol, uranic, severe infections such as typhoid fever, diphtheria, miliary tuberculosis, X-rays and radium, and when the bone marrow has been replaced, as in leukaemia and new growth.

## THE LEUKÆMIÆ (LEUCOCYÆ)

**Definition** (Edwards) <sup>1</sup>—The essential feature of leukæmia is a hyperplasia of certain reticulo-endothelial cells throughout the body, with differentiation towards one or other type of leucocyte.

In children leukæmia is always acute, and never chronic, as judged by adult standards. Gittens<sup>2</sup> (14 cases) showed 11 lymphatic, 2 myelocytic, and 1 monocytic. Newns<sup>3</sup> over a period of 11 years, at Great Ormond Street, recorded 56 with 50 acute lymphatic leukæmia, 5 monocytic and 1 myelocytic.

**Ætiology.** *Infective theory*—Because of the acute onset in some cases, with fever and ulceration of the throat it has been thought to be infective in origin. No proved cases of contact infection are known. Leukæmic mothers have given birth to healthy babies, and leukæmic babies have had healthy mothers. No organisms have been obtained from blood culture but certainly acute infections may produce a blood picture very like that of leukæmia.

*Neoplastic or new growth theory*—In animals, particularly mice, leukæmia may be transferred from one to another by intravenous inoculation. A subcutaneous injection of leukæmic blood will produce a localized infiltrating tumour. It is, therefore, in many ways closely allied to cancer in its mode of action.

**Age.**—It occurs between 2 and 5 years of age, and the average of 29 cases (Court and Edward) was 4½ years. On the other hand, infants have been born with the disease, and it may be seen in much older children and young adults.

**Symptoms.**—Instead of dividing the leukæmias into (a) acute lymphatic leukæmia, (b) acute myelocytic leukæmia, and (c) acute monocytic leukæmia, it is proposed to consider them all together. The reason for this is that the clinical picture is the same in all three.

**Onset**—This may be extremely acute, with fever, ulceration of the mouth and purpura. Accompanying these, there may be pains in the limbs, almost like rheumatism, and malaise.

In other cases, hæmorrhage may be the first symptom. Much bleeding from the gums or nose, stomach or bowel, may follow the removal of a tooth or some other minor operation. Bruising and purpura are features and retinal hæmorrhages occur.

The onset in most cases is insidious. There is loss of energy, lack of appetite and increasing pallor, with shortness of breath. Some enlargement of the glands, particularly of the glands in the neck, is usually present. Occasionally the salivary glands are much enlarged. The limb pains may be so severe that the parents suspect rheumatism.

**Course of the disease.**—This is usually rapid, and lasts a few weeks only. Not more than 3 remissions.

**Physical signs**—As the blood platelets fall toward the critical level of about 40,000 per c mm., hæmorrhage and purpura, with bruising, are

<sup>1</sup> D. G. H. Edwards. *Leukæmia in Children*. *Chin. Jour.*, March 1932, xviii, 190.

<sup>2</sup> B. Gittens and J. C. Hawksley. *Jour. Path. Bact.*, 1935, xxxvi, 115.

<sup>3</sup> O. H. Newns and A. G. Siguy. *Proc. Roy. Soc. Med.*, 1938, xxxi, 864.



more common. Occasionally there is ulceration of the throat and bleeding from the gums.

*Glandular enlargement* is widespread, if present. It is not confined to one type of leukaemia only. The spleen is palpable in 80 per cent of the cases and greatly enlarged in 25 per cent (Court and Edward 1939).

*X rays of the long bones* show patchy absorption of the cancellous bone, or diminution or thickening of the compact bone, due to the proliferation in the bone marrow. Occasionally the periosteum is raised due to leukaemic deposits.

*Blood changes—Leucocyte count*—In a series of cases the average was 70 000 per cubic millimetre. It may be as high as 200 000 or as low as 1 000 (leukanæmia). Usually it rises before death.

(a) In the lymphatic type the cells are lymphocytes or lymphoblasts and it is rare to have less than 90 per cent of this type of cell.

(b) In monocytic leukaemia there may be monoblasts as well as monocytes. In addition there are some lymphocytes and a number of other cells of the granular type. In a typical case there would be perhaps 60 per cent of the monocytic cells and their precursors. The diagnosis is made much easier by using a supra vital stain for the blood film.

(c) In myelocytic leukaemia which is rare there is a mixture of myeloblasts, myelocytes, polymorphonuclears, lymphocytes and monocytes.

A progressive anaemia in which the red cells and hæmoglobin fall together leaving the colour index at approximately 1 (orthochromic type) occurs along with the rest of the blood picture.

The platelets are reduced almost from the beginning of the disease.

*Pathology*—The glands and all the other organs of the body are packed with the cells corresponding to the type of response called up by the disease, i.e., lymphocytes, monocytes or myelocytes. The kidneys, liver, lungs, bone marrow and spleen are infiltrated. Rarer sites are the retina and skin. Subperiosteal nodes have been described. Sternal puncture will show the type of cell which is being manufactured in response to the disease. Sternal puncture should be done in all doubtful cases.

Biopsy of a gland will also make a definite diagnosis possible.

### Differential diagnosis.

1. Is it a case of agranulocytosis due to an infection? In such a case, the platelets are not reduced.

2. Purpura simplex shows no reduction of platelets.

3. Thrombocytopenic purpura, although showing a reduction of platelets shows no leukæmic blood count.

4. Scurvy shows no blood changes.

5. Hodgkin's disease shows no blood changes. The glands are in groups in the neck or abdomen and there is no widespread enlargement. These glands are soft and discrete. Biopsy may be required to clinch the diagnosis.

6. Tuberculous and lympho-sarcomatous glands may be differentiated by biopsy, and a blood count is helpful.

7. Glandular fever shows no anaemia and no reduction of the platelet count.

8 In agranulocytic angina there is no anæmia or reduction of the platelets

9 Rheumatism resembles this disease only in the vague limb pains and a cardiac bruit but a blood count differentiates at once

10 Whooping cough may show a lymphocytosis but there is no anæmia nor are there lymphoblasts

11 Rare conditions such as Allers Schönberg disease Cooley's anæmia and von Jaksch's anæmia are ruled out by the course of the disease

**Prognosis and treatment**—In all cases the subjects of leukaemia die In 29 cases the average duration of life was 10 weeks with the shortest 10 days and the longest 10 months (Court and Edward 1939) \*

X ray therapy may reduce the glands for the time being but it often hurries on the end Deep X rays to the mediastinum are called for when the breathing is embarrassed and there tends to be no recurrence of the disease in the treated site (Cooke 1932) †

Iron and arsenic tonics do not appear to be much help Pentnuchatide 10 c cm hypodermically for several days may produce a temporary remission in some cases of lymphatic leukaemia and is worth a trial

## DISEASES OF THE RETICULO-ENDOTHELIAL SYSTEM

### HODGKIN'S DISEASE (LYMPHADENOMA)

**Definition**—A fatal disease characterized by a painless progressive enlargement of the lymphoid tissue of the body in the glands spleen and other organs associated with progressive anæmia (Whitby and Britton)

**Ætiology**—The cause of the disease is not known It is probably a reticulo-endothelial new growth although it is thought by some to be a granulomatous infection akin to tuberculosis It may be found at any age but is rare below 10 years and is commoner in males than females

**Gordon's test** which consists of inoculating rabbits with an emulsion of a gland from a case of lymphadenoma and producing encephalitis has been shown to be fallacious

It depends solely on the presence of eosinophils in the material injected and has nothing to do with the disease itself Eosinophils are present in 70 per cent of glands of Hodgkin's disease and in the same percentage of cases the Gordon test is positive (McCausland) ‡

**Pathology**—The glands are enlarged and discrete particularly the neck group The glands of the abdomen and axilla may also be enlarged The spleen is greatly enlarged and is very hard and has been termed a hard bake spleen On section patches of whitish lymphadenomatous tissue are seen scattered over the organ Both the bone-marrow and the liver show nodules of this tissue The lungs kidneys and intestines are also affected

**Microscopically** the characteristic picture is an increase of the reticulo-endothelial cells which replace the lymphoid tissue There are

\* D G F F Iwatts and D Court, Monocytic Leukæmia in Children *Arch Dis Child* No. 2 1939

† 231

‡ J V Cooke, Mediastinal Tumour in Acute Leukæmia *Arch Jour. & Child* 1934 xlv 11 52

§ D J M McCausland, Hodgkin's Disease in Children *Arch Dis Child* 1941 xvi 69

multi nucleated giant cells, the nuclei being centrally placed. Usually, there are many eosinophil cells. Towards the end progressive fibrosis takes place, and little beyond fibrosis tissue is found in some of the organs.

**Blood**—A progressive hypochromic anemia is present. The leucocytes may be normal, increased, or decreased, but there is usually a leucopenia. Fifteen per cent show an eosinophilia (Whitby). The blood platelets are normal until the end, and the coagulation and bleeding time are also normal.

**Clinical picture.**—The onset is gradual, with enlargement of a group of glands, usually on one side or other of the neck. Wasting, pallor and weakness follow. The temperature may be high and continuous or remittent, or of the Pel-Ebstein type. The latter is seen in the abdominal type of lymphadenoma. In such a case the glands are largely confined to the abdomen, with much splenic enlargement. The temperature slowly climbs for several days, then slowly subsides and remains down for approximately a week, and then once more repeats the rising undulations. With each rise in temperature the symptoms grow worse, but subside again along with the temperature. There are pains in the bones, and pressure symptoms from the glands pressing on the trachea and bronchi, which may require surgical interference. Collapse of the lung and pleural effusion may be present (Figs 83, 84).



Fig. 32. Lymphadenoma in a boy aged 10, showing massive glands in the neck (By courtesy of Dr. Hedd & Cameron.)

#### Differential diagnosis :

- 1 Tuberculosis must be excluded. Generally, enlarged tuberculous glands go on to abscess formation, but lymphadenomatous glands usually remain discrete. The temperature is irregular, and not characteristic of lymphadenoma. A Mantoux or tuberculin patch test should be done, and the removal of a gland makes differentiation easy.
- 2 Leukemia is excluded by the blood picture.
- 3 Congenital syphilis is excluded by the Wassermann reaction.
- 4 In chronic septicemia, with generalized glandular and splenic enlargement, abscesses are formed.
- 5 Miliary tuberculosis and typhoid fever are differentiated by the rapid course of the first, and by the Widal test in the second.

**Prognosis.**—All cases of lymphadenoma end in death the duration of life being from 1 to 6 years.

**Treatment.**—X-rays or radium to the spleen and glands, particularly if administered by an expert, may alleviate symptoms for the time being.

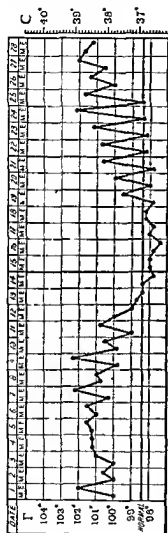


Fig. 33.—Example of intermittent temperature in lymphadenoma (Pel-Ebstein type)

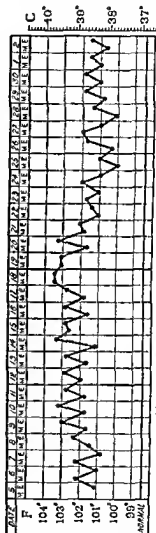


Fig. 34.—Example of continuous temperature in lymphadenoma

Iron and arsenic have some reputation, but cannot be said to be much use. Splenectomy has been performed, with no great benefit. Blood transfusion is definitely of value. If the pressure symptoms are great, causing pressure on the common bile duct, with jaundice, or pressure on the bowel, or

dislocation of the trachea or bronchus, operative treatment may have to be resorted to. All such treatment is merely palliative.

### GLANDULAR FEVER (INFECTIOUS MONONUCLEOSIS)<sup>1</sup>

Glandular fever is an acute infectious disease characterized by enlargement of the lymphatic glands, changes in the blood cells especially mononucleosis, heterophil antibodies in the serum, and a uniformly favourable prognosis.

**Ætiology.**—The condition is commonest in children and may occur in epidemics. The cause is unknown and is probably a virus.

**Pathology.**—The infection involves the reticulo endothelial tissue, causing a hyperplasia.

**Mode of infection.**—Infection is brought about by direct contact. It is not highly infectious. School epidemics are common.

**Incubation period.**—This is 5 to 12 days. The patient should be isolated for 1 week after the temperature is normal.

**Clinical picture.**—The commonest age is from 6 to 15 years.

The onset may be insidious but is usually acute, the temperature rising to 101° or 104° and remaining up for a week or longer. There is considerable malaise. In a small proportion of cases meningeal symptoms have been described. The tonsils are enlarged and injected, and in a proportion of cases there is a pale or white membrane on them, which can be easily removed, but which may superficially resemble diphtheria. Conjunctivitis may be present. The spleen is usually palpable and enlarges with the glands. Very often these are not painful, but they may be definitely tender. The enlargement is chiefly of the neck glands, but also in the axilla and groin.

Rashes are said to occur in 15 per cent. of all cases and to be of a morbilliform or macular type. Cases have been described showing well marked jaundice with other symptoms suggesting hepatitis.

**Blood.**—There is a leucocytosis, from 8,000 to 25,000 per c. mm. or more. A mononucleosis up to 60 or 70 per cent. is usual.

**Heterophil antibodies (Paul Bunnell reaction).**—This is the ability of a serum to agglutinate and hemolyse sheep cells. In glandular fever this ability rises rapidly, and by the fourth day has risen from the normal 1 in 8 to 1 in 64 or 1 in 250.

The Wassermann test may also be temporarily positive.

**Diagnosis.**—The enlarged glands must be differentiated from

1 Tuberculous glands, mumps, septic glands and Hodgkin's disease, by an examination of the blood for the mononucleosis and heterophil agglutination.

2 Monocytic leukaemia may be differentiated chiefly by the absence of the Paul Bunnell reaction, and the course of the disease.

**Treatment.**—This is symptomatic. Convalescent serum is said to be efficient. Aspirin, to relieve the headache, mouth washes, gargles, and

<sup>1</sup> Kilham and A. J. Steinman, "Infectious Mononucleosis," *Lancet*, Oct. 1<sup>st</sup> 1941, II, 437. H. E. T. Ian & M. L. B. Kilham, "Infectious Mononucleosis with Special Reference to Cerebral Complications," *Am. Jour. Dis. Child.* 1941, LII, 1111.

throat paints are indicated. The bowels should be kept well open and the diet should be as full as the patient is able to take while keeping a moderately clean tongue.

During convalescence, which may be prolonged, cod-liver oil and malt, and some iron preparation (*see p. 178*) should be given, a week of each in rotation.

**Summary of a typical case, seen by the author at the Hospital for Sick Children, Great Ormond Street, London, W.C.1**

Male, 6 years 9 months. On March 6, 1939, complaint of pain on moving his neck, and his mother noticed a swelling of the glands in the neck. He was otherwise quite well, ate well, and had no sore throat or diarrhoea, and did not vomit. There were three other children in the family and none of them had even a cough or cold.

On examination he was pale and ill looking. There were big red tonsils, the tongue was clean, there was a mass of enlarged cervical glands on each side which were not tender. The glands in the axillæ and groin were small but palpable. The liver was palpable, the spleen enlarged three finger breadths. Nothing abnormal was found in any other system.

**Investigations**—Mantoux negative. Sedimentation rate 13 mm in one hour (normal 9 to 10 mm). Widal and Brucella agglutinations negative. Wassermann a weak positive. Heterophil agglutination, Paul Bunnell. March 13, positive, 1 in 128, April 1, positive, 1 in 64 (normal 1 in 8).

**Blood count on admission, March 12**—

RBC	3,250,000 per c mm
Hb	65 per cent
CI	1
WBC	9,600 per c mm
Polys	10 per cent
Lymphs	90 "
Monos	8 "
Eosin	2 "

**Highest count**—

WBC	16,300 per c mm—87 per cent non granular cells
-----	--

**Typical count**—

WBC	11,300 per c mm
Polys	15 per cent
Small Lymphs	32 "
Monos	23 "
Atypical cells	28 "

Supravital staining shows these cells to be immature monocytes, stained with Leishman's stain, they are big cells with a slightly bean-shaped nucleus, and a big blue grey cytoplasm, with darker granules.

Discharged with a normal count on April 1. Spleen and glands subsided.

## DISEASES OF LIPOID METABOLISM AFFECTING THE RETICULO-ENDOTHELIAL SYSTEM (LIPIDOSES)

(a) **Gaucher's disease**: Substance deposited, kerafin.

**Definition**.—A rare disease which is not hereditary, but congenital and familial, characterized by an enlarged spleen and liver (both organs contain Gaucher's cells), leucopenia, and a hypochromic anemia.

**Sex**.—Seven females to one male, mainly Jewish.

**Age**.—May be recognized in infancy.

**Pathology**.—The spleen is enlarged and fibrotic and contains large Gaucher cells (20 by 10  $\mu$ ). These have small round nuclei, and are distended with lipoid material. The cells are probably reticulo-endothelial cells, and are found in the liver, bones, bone-marrow and lymphatic glands.

\* J. N. M. Chalmers: 'Gaucher's Disease. Diagnosis by Sternal Puncture and Improvement following Splenectomy,' *Arch. Dis. Child.*, 1910, **xv**, 239.

**Ætiology**—This is an inborn error of metabolism. The substance deposited is *kerasin*, which resembles *cerebrosides*.

**Symptoms**—Enlarged spleen is the first sign. The liver enlarges later. A yellowish pigment of the skin is present. There is a hypochromic anaemia, and occasionally spinal and bony deformities.

**Prognosis**—These cases live many years but may die of intercurrent infection, or anaemia.

**Diagnosis** can be made by puncture of the spleen or sternum.

**Treatment**—Symptomatic. Splenectomy may be indicated.

(f) **Niemann-Pick's disease**—Substance deposited *lecithin*.

**Definition**—An inborn error of metabolism resulting in widespread deposition of *lecithin*, with production of splenomegaly and other manifestations. Mainly in Jews.

**Sex**—Females largely.

**Onset**—Starts in infancy. It is congenital, familial but not hereditary.

**Pathology**—The cells are known as *foam cells*, and these are reticulo-endothelial cells distended with lipid material mainly *lecithin*. The whole body is involved, including the nervous system.

**Clinical picture**—In infancy the spleen, liver and glands become enlarged. The skin is yellow, the child wastes and ascites develops. There is also anaemia. The lesion of the nervous system is like that of Tay Sachs disease (amaurotic family idiocy see p. 200).

**Blood**—Leucopenia with raised cholesterol and leucocytosis.

**Prognosis**—Death takes place by two years of age.

**Diagnosis**—Splenic puncture.

(c) **Schuller-Christian's disease**: Substance deposited *cholesterol*.

**Definition**—An inborn error of lipid metabolism resulting in deposition of *cholesterol*, especially in the bones, with formation of granulomatous tissue. Mainly in Jews.

**Sex**—Males.

**Onset**—Early childhood, congenital and familial.

**Pathology**—There is a deposition of *cholesterol* forming *foam cells* in the reticulo-endothelial cells.

Skull is chiefly affected. The bone may be perforated by the deposits or the orbit may be encroached on, producing exophthalmos. The sinuses or the pituitary may be pressed on.

**Symptoms**—Bony defects, exophthalmos, diabetes insipidus and discolouration of the skin with xanthomatous, enlarged spleen, and hypercholesterolaemia.

**Prognosis**—The patient lives twenty years or more.

**Diagnosis**—X ray of bones and sternal puncture.

#### DISORDER OF GLYCOGENIC METABOLISM

##### GLYCOGEN STORAGE DISEASES

**Definition**—An inborn error of metabolism resulting in abnormal disposition of glycogen in the tissues, especially in the liver.

**Sex**—Both sexes.

**Onset**—Infancy. It may be congenital and familial and may be inherited as a Mendelian recessive.

**Pathology**—The liver is much enlarged and glycogen is deposited in excess in the liver, kidney and heart cells.

**Blood**—Both the glycogen and cholesterol are high.

**Blood sugar**—The fasting level is low, sugar tolerance test often shows no rise. After an injection of adrenalin there may be no rise.

**Urine**—Ketonuria.

**Clinical picture**—Enlargement of liver in infancy and childhood. This may become enormous. There is a lack of development and at the child remains infantile. Hypoglycaemic convulsions may occur.

**Treatment**—Not much effective.

## CHAPTER XI

### ANOMALIES OF THE GENITO-URINARY SYSTEM

A CAREFUL general examination of the child should be made for any points which might confirm or refute disease of this system

1 The blood pressure should be taken. It should be

Infants, 75 to 85 mm of mercury (systolic)

Aged 10 years, 90 to 100 mm " " "

Above 10 years, 100 to 120 mm " " "

2 Oedema should be carefully looked for in the limbs and dependent parts of the body

3 The heart should be examined for murmur, or other evidence of left-sided cardiac hypertrophy

4 Congenital abnormalities of the genitalia must be excluded. Make certain that the male or female urethra opens in the normal position

#### THE NORMAL URINE

Collection of specimen (a) *Males*—The cleansed penis should be placed inside a sterile test tube or bottle, which is kept in position with strapping

(b) *Females*—In suspected cases in the female, it is better to catheterize, as this is relatively simple. Failing this, in a tiny infant, the wrist of a boiled rubber glove should be fastened with adhesive tape over the vulva, and the urine collected directly into the fingers of the glove. Where a bacteriological examination is required from a female child, as in pyelitis, a catheter specimen should be obtained

Quantity of urine—About two thirds of the fluid intake is passed per day as urine

At 2 years the output is 15 ounces

4 " " 20 "

7 " " 30 "

12 " " 40 "

Micturition—In the *small infant* the urine is passed about every half hour. Control is rapidly acquired

At 6 months of age the infant is often dry when picked up to be fed if it has been placed on the chamber after the previous feed

At one year the child may be able to retain its urine for a period of 5 or 6 hours during the night, and 3 hours during the day

At two and a half years the normal and properly trained child should pass its water only on waking, after breakfast, at dinner time, after tea, and at bed time

Testing the urine.—A routine examination should be made in all cases for the following



**Albumin**—Heat and add acetic acid. If albumin is present, the urine should always be examined microscopically. Starch granules used as dusting powder, if present in the urine, cause a heavy cloud of what appears to be albumin on boiling.

**Globulin**—Is tested for by adding acetic acid to urine which has not been heated. Globulin is present in orthostatic albuminuria.

**Protein**—In each 12 hours, an individual excretes from 5 to 90 mgm of protein, but 95 per cent of children excrete less than 55 mgm which should be looked upon as the upper limit of normal (Snook<sup>1</sup>).

**Sugar**—Fehling's or Benedict's solution should be used.

**Acetone**—This is tested for by the Rother's test, performed as follows: to 1 c cm of urine add a crystal or two of sodium nitroprusside. Shake until dissolved then add ammonium sulphate and a few drops of strong ammonia. If the test is positive the urine assumes a deep purple colour.

**Diabetic Acid**—This is tested for with *Liquor ferri perchlor*. A positive test will result, however, if the patient has been taking salicylates.

**Pus or Blood**—Either of these is best confirmed or excluded by the microscope.

**Casts**—In each 12 hours, according to Snook a child should excrete up to 29,000 urinary casts, 95 per cent of children excrete less than 9,000 casts which are hyaline and granular in type. Above this number should be looked upon as abnormal.

**Red Blood Corpuscles**—In each 12 hours, according to Snook a child may excrete up to 600,000 red cells in the urine but above 600,000 should be looked on with suspicion.

**White Blood Corpuscles**—A normal child excretes one million and possibly as much as 2½ million white blood corpuscles in 12 hours. Above one million should be looked upon with suspicion (W. W. Payne).

### RENAL FUNCTION TESTS

**Urea-concentration test**—In the normal individual the urinary output contains about 4 per cent. of urea.

Where the kidney is damaged the concentration is reduced and may be as low as 0.5 per cent. The technique of this test is as follows: 10 grammes of urea, suitably dissolved and flavoured are given say at 6 a.m. the urine having been collected immediately before. A specimen is obtained at hourly intervals for, say, four hours, and the urea content of the various specimens is ascertained by means of Doremus's ureometer, hypobromite solution being used.

**Blood-urea test.**—Normally the blood urea should fall between 25 and 40 mgm per cent.

Where the kidney is damaged the blood urea may rise to 200 or even 250 mgm per cent.

**Urea-clearance test.**—The normal range is expressed in children as from 85 per cent to 115 per cent.

This is usually done by the van Slyke method. This rather complicated test is based on the ability of the kidney to remove a known quantity of

<sup>1</sup>A. W. Snook, "Prognosis of Glomerular Nephritis in Childhood," *Amer Jour Dis Child* June, 1929, viii, 1173.

urea from a measured quantity of blood in a certain time. It appears to be the most delicate and efficient kidney function test. Figures outside these indicate some abnormality in the kidney function.

### ALBUMINURIA

Albumin may be found in the urine in minor infections as well as serious kidney infections. For instance accompanying infections such as diphtheria, whooping-cough and pneumonia, there is often a trace of albumin which disappears with the cessation of the disease. In infants transient albuminuria is common with severe dehydration or diarrhoea and vomiting. The severe albuminuria following scarlet fever or streptococcal sore throats and leading to true nephritis will be dealt with under that heading (see p 208). It is sufficient to say that albuminuria may or may not be significant but in all cases a thorough general examination of the patient should be made and its cause sought. The most common form of albuminuria is one which occurs quite apart from any apparent infection or organic disease namely, orthostatic albuminuria.

### ORTHOSTATIC OR POSTURAL ALBUMINURIA (PHYSIOLOGICAL ALBUMINURIA)

This appears most often in children between the ages of 10 and 15 when the child is growing rapidly and approaching puberty and is slightly more common in boys than girls. The specimen of urine obtained before rising in the morning is quite free from albumin. As the day goes on each specimen contains a larger and larger quantity until towards the evening an appreciable amount is found. On going to rest in the evening the urine once more becomes free. If the child remains in bed throughout the 24 hours the urine remains quite free showing that the albumin is due to being up and about or in other words to posture. In such urine the globulin can be precipitated by the addition of acetic acid, without heating. Serum albumin is also present and is precipitated by heating.

**Clinical picture**—The children who suffer from albuminuria are as a rule tall slim overgrown and pale with a tendency to faint easily and get too tired. They stand badly with the shoulders drooping and the pelvis pushed forward in fact the stance is that described under the nervous exhaustion child (p 265). Headaches are common, especially towards the end of the day and a variety of symptoms readily attributable to vaso motor instability may be present.

**Prognosis and treatment**.—Children invariably grow out of this complaint and no particular treatment is necessary for the albumin. Tonics and general toning up, with plenty of rest and good food, are all that is indicated.

### HEMATURIA

Hæmaturia may be due to

1 Renal causes—(a) In *nephritis* there will be albumin apart from that accounted for by the blood. The blood urea may be raised.

(b) *Calculus* is accompanied by pain which radiates towards the bladder and even to the tip of the penis or there may be aching in the groin.

An X ray of the renal tract and ureters will often show the stone. Such calculi are sometimes found in cases of abdominal tuberculosis or spinal caries, where the child has been compelled to remain still in bed on his back for some time. They are often accompanied by a urinary infection.

(c) *New growths*—Hæmaturia may be the earliest symptom of renal sarcoma or Wilm's tumour (see p. 215). An X ray after intravenous uroselectan, or retrograde pyelogram will demonstrate the size of the kidney. Palpation of the kidney under an anæsthetic may be necessary.

(d) *Scurvy*—A general examination will show the condition of the infant's gums, tenderness of the limbs on movement and a shortage of vitamin C in the diet.

(e) *Renal tuberculosis*—Usually this condition is painless and tuberculous infection may be found elsewhere in the body. An examination of the sediment from the urine may show tubercle bacilli or when injected into a guinea pig it may produce the disease.

(f) *Blood diseases*—In the leukoses and hæmorrhagic diseases, an examination of the blood will confirm the diagnosis. The bleeding may be slight or very extensive, as in hæmorrhagic nephritis. The child should be examined for purpura, and the size of the spleen and glands noted.

(g) *Oxaluria* may produce slight bleeding. A careful examination of the urine for crystals will reveal them.

(h) *Trauma* may be indicated by a history of a fall and some external bruising.

(i) *Blackwater fever* will be confirmed by repeated blood examinations and the history.

2 *Ureter, bladder and urethral causes*—Hæmaturia may be caused by—

(a) *Papilloma of the bladder*—Bleeding will be profuse, and some of the papilloma may be seen microscopically but a cystoscopy will be necessary to confirm the diagnosis.

(b) *Urethral or meatal ulcer*—This is the commonest cause in children under 3 years of age (see p. 206).

(c) *In pyelo cystitis*, blood as well as pus is present.

### CONGENITAL ABNORMALITIES

These are comparatively rare, being found in from 2 to 4 per cent. of all autopsies. Many are now discovered in life since the introduction of intravenous uroselectan with X rays, and after retrograde pyelogram. The frequent use of the cystoscope has also revealed further abnormalities of the bladder.

#### 1 ABNORMALITIES OF THE KIDNEY

(a) *Congenital cystic kidney*.—A rare condition in which the kidneys may be so large as to obstruct labour. Usually, they can be felt on palpation but they may be unsuspected. The symptoms are those of interstitial nephritis (see p. 217). The kidneys are loculated and the kidney substance almost destroyed. This is a developmental error.

(b) *Congenital absence of one kidney and ureter, or fusion of the kidneys (horse-shoe kidney)*.—These are discovered usually by chance.

(c) **Pelvic kidney** may be palpated, and may or may not give rise to symptoms

## 2 ABNORMALITIES OF THE UPTERS —

(a) A duplication of the ureters will be discovered by cystoscopy and after uroselectan

(b) Aberrant renal vessels may link the ureters and be a cause of congenital hydronephrosis

(c) Strictures of the ureter are the commonest cause of hydronephrosis or pyonephrosis. Intermittent pain may be caused. More commonly, they are discovered on examination of the urinary tract in a case of chronic pyuria and are the cause of the failure to react to treatment

## 3 ABNORMALITIES OF THE BLADDER, URETHRA AND THE GENITAL ORGANS —

(a) **Extroversion of the bladder**—This is due to an arrested development of the ventral wall of the urogenital canal. The anterior wall of the bladder and the corresponding portion of the abdominal wall are absent and the symphysis pubis fails to fuse. In males the penis is deformed and epispadias results. In the female the labia are separated and the cleft urethra continues into the exposed bladder. The bladder is seen as a red protrusion in the pubic region. The urine runs away continually, eczema of the skin results. A large number of infants die in the first few years of life.

**Treatment**—Plastic operations are recommended

(b) **Epispadias**—The urethra opens on the dorsum of the penis

(c) **Hypospadias**—The urethra may open at the level of the glans, the body of the penis or the junction of the penis and scrotum

**Treatment**—Plastic operations for both conditions are recommended

(d) **Pinpoint meatus**—This is common and may cause pain on micturition especially in summer time when the urine is concentrated

**Treatment**—Opening the meatus and keeping it open with a glass rod dipped in liquid paraffin

(e) **Undescended testicle (Cryptorchidism)** See p. 374

(f) **Phimosis**—If the prepuce in a newly born infant is retractable only with difficulty the question of operation then arises. When there is sufficient room for the infant to pass urine and the foreskin can be retracted even with pressure there is no real need for operation. On the other hand for the sake of cleanliness and to obviate the possibility of early masturbation it is undoubtedly in the author's opinion an advantage to circumcise

(g) **Atresia of the vulva (Adhesions)**—This condition is comparatively common. The labia minora are joined by a thin layer of mucous membrane so that the urethra and vaginal opening are covered up. The treatment is to break down these adhesions with a probe and place in the opening a pledget of cotton wool, smeared with vaseline

(h) **Prolapse of the bladder, diverticulum of the bladder, and prolapse of the uterus**, are all extremely rare and easily recognized

(i) **Congenital valvular obstruction of the urethra**—There is a history that the child has never passed urine properly. Later as a result of back pressure a dilated bladder, ureters and kidney will occur with destruction of the kidney substance. The child remains infantile and dies of uræmia. An examination of the posterior urethra will show a partial diaphragm or fold of mucous membrane such as is found in a vein. A catheter passes readily into the bladder, but cannot be pushed outward from it.

**Treatment**—Rupture and removal of the valve by the retrograde passage of a bougie from the bladder has been successful when the condition has been recognized early enough.



Fig. 35.—Pyelonephrosis showing dilated pelvis and tortuous ureter.

## INFECTIONS OF THE URINARY TRACT

### PYELITIS PYELONEPHRITIS AND PYELOCYSTITIS

This disease is much commoner than is at present realized particularly in the neo-natal period. Craig (1935)<sup>2</sup> was able to collect 61 cases in the newly born.

<sup>2</sup> W. S. Craig. Urinary Infection occurring in the Neonatal Period, *Arch. Dis. Child.*, 10, p. 337.

**Sex-incidence**—It is almost as common in boys as in girls under one year 34 girls to 27 boys (Gorter). In a series reported by Griffen<sup>1</sup> there were 33 boys to 43 girls in the first 96 weeks of life. After the age of one year there are about three girls to one boy.

**Age-incidence**—About one third of the cases occur between birth and 6 months, one third between 6 and 12 months and the remaining third between 1 year and 18 years.

**Ætiology.**—There are two theories on the mode of infection—

1 *Ascending route*—The shortness of the urethra in the female and the high incidence of pyelitis in that sex led to the inference that the vulva became contaminated by faeces and that the organism (*B. coli* as a rule) passed directly into the bladder and then to the pelvis and substance of the kidneys. This is an attractive and very old theory, which receives very little experimental corroboration.

2 *Infection of the blood stream (hæmatogenous route)*—In the great majority of cases this is probably the mode of infection. The infant develops a *B. coli* septicæmia and the kidney becomes infected as a result.

Some most interesting experimental work on this subject has been done by Miss Lepper<sup>2</sup>. She has shown that if a strain of *B. coli* which has produced pyelitis in an infant is injected into the ear vein of a rabbit and at the same time either the ureter or the renal vein is clamped off so that the urine is dammed back into the pelvis of the kidney or the kidney becomes congested then pyelitis occurs. Even if the clamping off is very transitory the kidney is nevertheless selected as the site of infection from the whole body.

It has been known for many years that pyelitis is common where there has been malformation of the urinary tract which dams back the free flow of urine. Blinked ureters giving rise to hydronephrosis, are present in many cases which subsequently develop pyelitis. This same principle of clamping off the ureter or obstruction to the outflow of urine is seen during pregnancy where the infant's head at the brim of the pelvis pressing on the ureters accompanied by some constipation may cause pyelitis.

It is suggested then that the organism gains entrance from the bowel directly into the blood stream and settles in a congested kidney, the congestion of the kidney being due either to venous stasis or urinary stasis from obstruction of the urinary tract.

**Pathology**—Post mortem examinations of simple pyelitis are practically unknown though the pathological appearances are occasionally seen at autopsy by accident. In other words simple pyelitis is seldom fatal. What is seen at post mortem examination is pyelo-nephritis.

In simple pyelitis there is a mild inflammation of the pelvis of the kidney. In the more severe cases however there is always a pyelo-nephritis. The glomeruli, tubules and interstitial tissue are all involved. Round cell infiltration is seen microscopically. This disease therefore is a nephro-pyelo-cystitis.

<sup>1</sup> Mary Griff. "Pyelonephritis in Infancy and Childhood: Its Bacteriology and Pathology." *Arch. Ped. Child.*, 1934 ix, 105.

<sup>2</sup> Elizabeth Lepper. *Journal of A. Bact.*, 1921 xxii 19.

**The organism** is most commonly the *Bacillus coli*. In Griffen's series of 87 cases, the distribution was —

Pure <i>Bacillus coli</i>	59 per cent
<i>Bacillus coli</i> with mixed infection	26
Streptococci	13
Staphylococci	2

*Bacillus proteus* and other organisms are occasionally found

**Symptoms**—The onset may be sudden and commence with pallor collapse, and a rigor. This however occurs in only a minor proportion of the cases. In the majority the onset is ushered in by fever with a temperature of 103° or 104° F. and some irritability. The stools may be frequent, and there is vomiting and refusal of food. The urine may be scanty, and may stain the napkins. Sweating and restlessness with thirst and irritability, are usually present.

For purposes of description the disease may be divided into the following forms —

**Cerebral type**—In some cases there is head retraction, squint and screaming. Occasionally, there are convulsions which suggest a severe infection, and in the newly born are of grave prognostic significance (Craig).

**Respiratory type**—In other cases there is high fever, rapid grunting respiration, and drowsiness, and there appear clinically to be respiratory infections without localizing signs.

**Abdominal type**—In infants the majority have abdominal symptoms. The abdomen is tumid and tender and there may be marked diarrhoea and some vomiting.

**Diagnosis**—This can only be made by a careful microscopical examination of the urine. In males, the urine should be collected in a clean test tube or bottle, or the infant should be catheterized forthwith. In females the method described on p. 194 should be used or the infant should be catheterized.

**Urine**—In the normal infant or child not more than an occasional pus cell should be seen in a low power examination of a shaken up specimen of urine. In cases of pyelitis, however there are as a rule from 5 to 100 or more pus cells per low power field in a shaken up specimen. Occasionally, at the commencement of the illness a specimen may be found with many organisms but few or no pus cells. There may be a varying amount of blood. A profuse growth of organisms will be found on culture. Albumin is always present, but there may only be a trace in some cases.

**Course**—A child with a straightforward pyelitis is much improved in three days, and feels quite well in one week. The urine may take two or three weeks to become quite clear, however. In severe cases the disease lasts much longer.

**Prognosis** In newly born infants—There was a mortality of 15 per cent in Craig's series of 61 cases, during the neonatal period.

In older infants and young children the mortality is much lower. Where

the kidney is much involved, uræmia, convulsions and death may occur. In a certain proportion the symptoms subside, however, and chronic pyuria persists, with recurrent bouts of acute pyelitis. Probably some congenital malformation of the urinary tract is present in a high proportion of such cases.

In older children the prominent symptoms are rigors, headache, vomiting and abdominal pain. This latter may be merely a dull, aching pain in the loins, with frequency of micturition, but is occasionally an acute abdominal pain, severe enough to simulate appendicitis.

In a few cases, intermittent pyuria, with abdominal pain and some fever, occur. Careful examination and culture between attacks show the urine to be normal and sterile, and a retrograde pyelogram shows a normal urinary tract. It is possible that these most difficult and puzzling cases are due to a neuro-muscular spasm of the ureters, and it is claimed that presacral sympathectomy is useful in treatment (Higgins).<sup>1</sup>

**Special investigations.**—In all cases where the urine has not cleared up in four to six weeks an investigation of the urinary tract should be undertaken. In males, this should be as far as possible by intravenous uroselectan (5 to 10 c cm) and X-rays. In females, a retrograde pyelogram with an opaque salt, such as sodium iodide (18.5 per cent.), is most useful.

### Treatment.

**1. Sulphonamide group.**—This treatment (for dosage, see p. 388) should be given for three or four days, then discontinued and then recommenced if necessary. It has the advantage of acting in an alkaline medium. On such treatment, in Fleming's series<sup>2</sup> of cases, 75 per cent. of the urines were sterilized where there was no organic lesion. Only 43 per cent. of the urines were sterilized, however, where an organic lesion existed.

**2. Hexamine (Urotropin)** depends for its action on the liberation of formaldehyde. This is a safe drug. A convenient mixture is:—

Hexamine, 2 grains  
Glycerine, 2 minims  
Water to 60 minims

This may be given to an infant each four hours. If the urine is not sufficiently acid (attaining a pH of 5 or 5.5), this treatment will be a failure. The urine is tested with methyl red and if sufficiently acid, it turns pink or red—if insufficiently acid it turns yellow. In that case some of the following mixture should be given in addition to the hexamine:—

Ammon. phosph., 7½ grains  
Liq. ext. of liquorice 6 minims  
Acid syrup, 15 minims  
Water, to 60 minims

Commence by giving 60 minims three times daily, and increase or decrease the dose as necessary.

**3. Mandelic acid.**<sup>3</sup>—This acts by liberating ketone bodies. It is best

<sup>1</sup> T. T. Higgins, "Genito-Urinary Diseases," *Practitioner*, April 1939, vol. 437.

<sup>2</sup> G. D. Fleming, "Subclinical Infection in Children," *Arch. Dis. Child.*, 1932, xiv, 276.

<sup>3</sup> G. H. News and R. Wilson, "Mandelic Acid in the Treatment of Pyelitis in Childhood," *Lancet*, Nov. 7, 1936, ii, 1987. M. L. Rosenheim, *Lancet*, 1935, i, 1022, and ii, 711.



GIVEN AS —

Ammon mandelate 26 grains  
 Liq ext of liquorice  $\frac{1}{2}$  minim  
 Flyr of glucose 1 lb,  $\frac{1}{2}$  minim  
 Water to 60 minims

If there is nausea an equivalent dose of calcium mandelate in chocolate covered granules is best given. The dose per day varies from 30 minims of the mixture eight hourly day and night to an infant up to 90 minims of the mixture six hourly day and night to a big child.

If the urine does not become sufficiently acid as tested with methyl red then the ammonium phosphate mixture quoted above should be given in sufficient quantities to get the urine strongly acid. Some restriction of fluid intake facilitates the cure. Alkalinizing foods, such as vegetables and fruit are best left out during this treatment.

4 Alkalis — Alkalis should be administered in doses sufficient to render the urine alkaline. It has been shown that once the urine is strongly alkaline to litmus *Bacillus coli* infections tend to clear up. The dose must be given at four hourly intervals, day and night to be of any real service and it takes from 10 days to 8 weeks as a minimum to effect a cure. The most suitable mixture is —

Pot cit 10 grains  
 Soda bicarb 10 grains  
 Syrup q s  
 Water to  $\frac{1}{2}$  oz

Two drachms each four hours day and night may be given to begin with. This dose may be increased or decreased.

No one drug will cure all the cases. Alkalis are unpleasant to take and tend to produce diarrhoea in infants. Hexamine is 75 per cent efficient and is pleasant to take. Mandelic acid is most unpleasant to take but is efficient except with streptococci and staphylococci. Sulphonamide is very efficient but not tolerated by all patients. It is best therefore, to change from one drug to another if difficulty is experienced.

Diet <sup>1</sup>—Formerly, a ketogenic diet was advocated but it is not now considered necessary.

The food should be reduced in quantity and all milk given well diluted. When treatment with alkali is being instituted alkalinizing foods, such as vegetables and fruit juice should be given freely but where hexamine and mandelic acid are given they should be withheld.

In the older child a light fever diet may be given and starchy food added to the milk to increase the caloric value. Plenty of sugar or glucose should be offered. The bowels should be kept carefully open with milk of magnesia preparations or a vegetable laxative such as cascara.

#### ENURESIS<sup>2</sup>

Before starting the treatment of enuresis it is absolutely essential that organic disease should be ruled out. The urine must be carefully examined to exclude pus which would suggest cystitis or pyelitis. Again a trace

<sup>1</sup> Pearl Summerfeldt and others, "Ketogenic Diet in Persistent Pyuria," *Brit Jour Child Dis* 1935 x 329

<sup>2</sup> Donald Paterson "Hints on the Treatment of Enuresis," *Clin Jour*, August 1937 vol 416

of albumin in a pale, low specific gravity urine suggests interstitial nephritis with polyuria. If congenital abnormalities elsewhere in the body are present an examination of the spine for spina bifida occulta should be made. Diabetes insipidus and diabetes mellitus must also be excluded.

Enuresis of the functional type is the one with which this section deals.

**Symptoms and diagnosis**—This condition is much commoner in boys than in girls, and in the great majority of cases a careful history reveals two facts: (1) that the mother or father usually the latter wetted the bed as a child beyond the normal period. (2) that the patient is passing his water too frequently during the daytime.

The inherited tendency to poor sphincter control and hyper excitability of the bladder reflex is the most important factor. Contributing factors are—

1. Frequency of micturition during the daytime so that the bladder holds only small quantities of urine without discomfort.

2. The general health of the child is below par. In considering the child from this standpoint two possibilities contributing to his ill health are—

(a) Chronic infections such as bad teeth and enlarged tonsils and adenoids.

(b) Physical exertion and mental over excitement (nervous strain). These children are often the nervous highly strung type, who enter into everything in life with great enthusiasm and tend to overtire themselves. They are too much on their legs and too active mentally. Frequently they are only children or the youngest in the family.

**Treatment and prognosis**—Most of the cases have completely recovered before puberty in fact after the age of eight very few are met. As the child gets older his nervous system tends to become more and more stable and the inherited tendency to poor sphincter control passes off—he is said to have outgrown it. This tendency cannot be directly affected by treatment but the contributing factors may be greatly modified by careful management.

The practitioner should investigate each case by getting the mother to keep a chart of the quantity of urine passed and the time it is passed from the moment of waking until bedtime. It will be found that the less frequently the urine is passed the larger the bladder will become. As the bladder becomes larger dry periods are obtainable at night.

1. The frequency of micturition must be checked.—This may be accomplished to a great extent voluntarily by the child itself. If he is very young emptying the bladder regularly at gradually lengthening periods will tend to distend it slowly so that it holds larger and larger quantities of urine. The value of this is that when the bladder is properly emptied before going to bed the child is tucked over the night period. To help in this belladonna is of the greatest service as it tends to cut off the nervous control of and therefore to relax, the wall of the bladder, rendering it less irritable. It then fills passively and contains a gradually increasing amount of urine. A suitable dose of belladonna for children of various ages will be found below.

2 Sepsis.—To improve the general health of the child sources of sepsis such as carious teeth and enlarged tonsils should be attended to.

3. Rest.—If the child has not begun to attend school it is well to employ him throughout the morning in some simple kindergarten lessons, so as to keep him off his legs and therefore rest him. Simple games will keep his mind employed and divert his attention to other things than emptying his bladder.

4 Drugs.—*Ephedrine hydrochloride* may be given at bedtime a quarter of a gram to a child of three to six years, and half a gram to an older child; this may not be a big enough dose, and more may be given if it is ineffective. It is often best to split the dose, giving one portion at bed time and one when lifted at 10 p.m. If, after a week's trial, there is no improvement the younger child's dose is increased by a quarter of a gram and that for the older by half a gram. This can be repeated until two or three grains are being taken by the older child, or until enuresis ceases.<sup>1</sup>

*Belladonna*.—To relax the wall of the bladder, 5 to 10 minims of tincture of belladonna should be given three times daily. It can be given alternate weeks for five or six courses.

**Psychological aspect.**—One of the most important aspects of this disability is the child's outlook towards his complaint. In most cases, and certainly in the most difficult cases, a too serious view is taken by the mother. The child is cautioned and urged to try to be dry. He is commended when successful and reprimanded when unsuccessful. He is impressed with the importance of overcoming his failing and with the fact that success or failure will make or mar his whole future. All this tends to place a conscious or subconscious strain on the child. An air of optimism about the whole matter must be carefully cultivated by those in charge. Indifference should be shown when he fails and praise when he succeeds. He should occupy a room by himself, so that he alone knows when he has succeeded or failed. No discussion by the family or any outsiders should be allowed.

To summarize, the treatment is training the child to hold his water during the day, improving the general health, removing septic foci and providing further physical and mental rest and interest. The psychological aspect must also be kept in mind.

### FREQUENCY OF MICTURITION

In infancy, the diet being mainly fluid and the quantity large,<sup>2</sup> the bladder must be emptied at frequent intervals. The normal infant passes water after each feed at least, sometimes two or three times between feeds, and several times during the night. By the age of 18 months, control of the bladder by day should be firmly established and by the age of 2½ years the child should also be dry at night. Frequency of micturition cannot be looked on as a pathological condition in the young infant, but as one necessitated by the diet, and by the small size of the bladder.

<sup>1</sup> *Lancet*, Oct. 7, 1929, p. 117.

<sup>2</sup> Donald Pederson, "Frequency of Micturition in Infancy and Childhood," *Practitioner*, June, 1926, cxvii, 618.

From birth onwards an attempt should be made to establish an association between the act of micturition and placing on the chamber (see p 11) so that unless he is placed there the child will make a voluntary effort to hold his urine, with consequent enlarging of the bladder and an increased ability to control the sphincter. Lack of training in this simple matter is much the commonest cause of frequency in young children, and thus leads very often to enuresis (see p 203). The percentage of bed wetters and children having frequency is many times higher among the poor than among the well-to-do.

Frequency may be associated with cystitis or stone in the bladder but both these conditions are rare in childhood. A careful examination of the urine should be made in every case. Strongly acid urine seems to act as an irritant with some children and a little bicarbonate of soda or potassium citrate will relieve this. The amount of urine should be carefully measured, to exclude diabetes or interstitial nephritis, as in both these conditions frequency is due to the quantity of urine passed.

Occasionally frequency is due to *habit*. The author has known cases where children have been in the habit of helping themselves to drinks at intervals throughout the day, with consequent frequency, when taken into hospital this habit has been broken and the frequency has rapidly disappeared.

In cold weather, especially when children are underdressed there is a strong tendency for an increased urinary output with frequency. In warm weather the reverse obtains.

Thirsting in some children is undoubtedly accompanied by bouts of frequency probably as a result of the child refusing solid food and taking in much larger quantities of fluid.

Nervous children often show spells of frequency between tea and bed time, or at any period of the day when they are under nervous tension or much in the limelight. Children going to school for the first time, or at a dancing class, or when being played or romped with, especially in the presence of strangers exhibit this tendency. The explanation undoubtedly is the child's fear of an accident. When removed from the exciting surroundings, the apprehension at once disappears and with it the frequency. In all cases of frequency, repeated careful examinations of the urine are necessary to exclude the possibility of organic disease.

The greatest possible help is obtained in such cases by giving a mixture containing 5 to 10 drops of tincture of belladonna three times daily, when the wall of the bladder tends to relax and the bladder to hold a larger and larger quantity of urine.

#### MEATAL ULCER

In male children who are still at the age when they cannot be kept dry both day and night or in those who are running about shortly after having been circumcised there is a tendency for an ulcer to form at the external meatus of the urethra. A crust forms and, when the urine is passed, this is torn off and remains bleeding, only to re-form before the next micturition. In such cases a great effort should be made to get the child to be dry both day and night. The local treatment consists in applying to the penis a piece of lint thickly smeared with vaseline, sewn in the pyjamas at night.

and in the underclothes by day, but kept in place during the whole 24 hours. A little tincture of belladonna is a great help, thereby checking frequency (see p 205). As a rule, there is an ammonia dermatitis which is keeping up the condition. (For treatment of this, see p 288.)

### PIN-POINT MEATUS

Pin point meatus is much commoner than is generally supposed. Although there is very little complaint that the urine cannot be passed, the stream is extremely small and the child has to exert pressure. In warm weather, with any concentration of the urinary salts there is a tendency to smarting, irritation or actual pain at the tip of the penis and micturition is dreaded. In such cases a meotomy is undoubtedly indicated and is a great advantage. In older children a pin point meatus may cause abdominal pain.

### PHIMOSIS

The question whether a child requires circumcision is always a debatable one. If there is a long and tight foreskin, so that the glans cannot be uncovered, stretching should be sufficient. In the author's opinion, however, this is not enough. Unless the greatest care is exercised, the foreskin becomes sodden with urine and inflamed and this inflammation leads to irritation and a tendency to attract attention to the penis, with resulting masturbation. Circumcision is better done early than when the child is older, as in the young infant, a few days or weeks old, extremely little upset is caused, and only slight anaesthesia is required.

When the foreskin is extremely tight, actual back pressure and interference with the passage of the urine may occur so that dilatation of the bladder and ureters is possible. There is no doubt that the circumcised child can be kept cleaner and is less likely to develop bad habits at an early age than the uncircumcised.

### BALANITIS

This is usually due to the presence of a long foreskin and lack of cleanliness. The swelling may be great, and there is pain on micturition. The treatment is bathing with boracic lotion or applying fomentations, later keeping the part dry with starch and zinc powder. To prevent a recurrence circumcision is advisable.

### HYDROCELE

There may be a hydrocele of the tunica vaginalis or of the cord. In all cases which persist for more than a few months, a hernia accompanying the hydrocele should be suspected. Spontaneous cure is common and certainly unless causing great inconvenience, nothing more than simple aspiration should be done until the child has acquired cleanly habits, say at the age of 2 years, when an operation for both hernia and hydrocele is advisable.

### VULVO-VAGINITIS

The frequency of this condition varies with the climate and the class, and is less in cold countries and among the well to do. About one-fourth of all cases are gonorrhoeal. It occurs most commonly between the ages of 2 and 5 years, but may occur at any age. It is most prevalent in summer.

**Gonorrhoeal and non-gonorrhoeal cases.**—Among the non-gonorrhoeal cases the chief source of infection is the feces, the vulva being infected especially by *B. coli* and the intestinal streptococci. Occasionally,

the diphtheria bacillus and pneumococci are found. Threadworms setting up an irritation about the anus and causing scratching of the anus and vulva are often responsible for the spread of the organisms.

Gonorrhoeal cases are always contracted from some other person and it is this type of case which tends to spread from child to child in an institution.

**Clinical picture**—The vulva is red and swollen and during the acute stage a thick purulent discharge can be seen covering the labia and appearing over the vaginal orifice. Pressure over the urethra may show a urethral discharge. There is pain on micturition in a proportion of cases. In the non-gonorrhoeal type the discharge tends to be less thick and watery and to have an offensive odour and pain on micturition is less common. Occasionally pneumococcal peritonitis which has been described on p. 112 complicates the non-gonorrhoeal type. In the gonorrhoeal type urethritis is common. Gonorrhoeal peritonitis or infection of the tubes is rare.

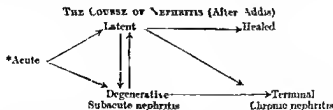
**Treatment**—Both in gonorrhoeal and non-gonorrhoeal vaginitis a course of one of the Sulpha drugs such as sulphadiazine or sulphamethazine (for dosage see p. 343) should be given. This may need to be repeated on two or three occasions. Local measures such as a sitz bath using warm water coloured pink with Condy's fluid are also indicated. A search for threadworms (see p. 394) shall be made.

In addition to a Sulpha drug treatment with oestrin<sup>1</sup> may in some cases be necessary. The object is to produce an epithelialization of the vaginal mucous membrane. The new squamous cells are washed out duly with a douche and in one to two weeks the thick discharge gives place to a thin watery non-gonorrhoeal discharge. The daily dose is 1 000 I.U. hypodermically or 5 000 units by the mouth. The duration of treatment is two to three weeks on the average.

The child should be kept off her legs and given as much rest as possible.

### NEPHRITIS

It is proposed that the terms acute hemorrhagic nephritis, acute focal nephritis, acute glomerular nephritis and parenchymatous nephritis should be dropped. No attempt is here made to classify nephritis according to anatomical or pathological lesions. The classification of Addison is adopted and the author thinks that this simplification will appeal to the student.



\* Meaning acute nephritis, edematous or non-edematous, excluding pyelonephritis, acute interstitial nephritis and acute embolic nephritis.

The chief distinguishing clinical characteristics of these conditions may be summarized as follows—

**1 Acute nephritis (first stage).—**Previously known as acute hæmorrhagic or glomerular nephritis

**Ætiology**—In 75 per cent of cases, the symptoms follow an acute upper respiratory infection. Tonsillitis with cervical adenitis, otitis media, mastoid disease, scarlet fever, and pneumonia are the precursors. In 25 per cent of cases the initial infection is so slight that it escapes notice altogether. *Streptococci pneumococci* or hæmolytic *staphylococci* have been recovered from the throat swabs.

**Age and sex**—It is equally common in boys and girls and the commonest age is between 3 and 10 years. But the cases are mainly found between 3 and 5 years.

**Symptomatology**—The onset of the disease is on an average 10 to 15 days after the infection, but it may start from one to twenty days after the initial illness. Much the commonest symptom is hæmaturia. The urine may suddenly become claret coloured or smoky brown. Some oedema, especially of the face or about the eyes, appears. There is often vomiting and abdominal pain, with headache and backache. In the more severe cases there is drowsiness.

Urinary symptoms such as frequency of micturition, enuresis and dysuria, also occur. There may be constipation or diarrhoea and increased thirst. Oedema may occasionally be marked and the dependent parts are most affected. Occasionally, the attack may be ushered in with a convulsion.

In about one-fourth of the cases the onset is insidious and were it not for the hæmaturia and perhaps slight oedema, nothing would be seen wrong with the child.

**Differential diagnosis**—A reference to the causes of hæmaturia on p. 196 will help the practitioner to exclude these in any given case.

The urine must be carefully examined under a microscope when red blood cells, white cells, casts and the absence of organisms will be noted. The blood urea will usually be found to be raised in the first few days. The abdominal pain may be severe enough to simulate appendicitis, and the frequency of micturition and dysuria confuse the case with one of pyelo cystitis. Where convulsions occur it may be thought to be the onset of meningitis, and lumbar puncture will exclude that diagnosis.

In cases presenting themselves for the first time with an insidious onset of gross oedema and gross albuminuria, in the absence of an acute infection, and without microscopic hæmaturia, the diagnosis of subacute nephritis is by far the most likely one.

**Special investigations**—A complete examination of the urine, especially by microscope, is essential. The blood urea will be found to be high, but does not help much in formulating a prognosis. The urea clearance and concentration tests are best not done at this stage. Except when there is much oedema, the blood cholesterol will be normal (100-200 mgm. per c.cm.) and therefore of no assistance in diagnosis.

**Pathology**—There is little doubt that the brunt of the infection falls on the glomeruli. The capillary loops of the tufts show necrosis in some instances. There is blood in the capsular spaces. Round cells may be seen near the affected glomeruli.

**Prognosis** —(a) The *immediate* mortality is less than 5 per cent. Signs which suggest danger are convulsions, broncho pneumonia, gastro-enteritis and a falling output of urine, together with a rising blood urea after the first week of the disease.

(b) *Remote*.—There is a complete recovery of about 40 per cent. The remainder may pass into the latent or second stage (see p 211) and be symptomless. They are able to undertake sport, and resume ordinary life but a microscopical examination of the urine will show that the renal process has not healed. Such cases may develop hypertension and renal failure later.

**Treatment** *Rest in bed*—The child should be put to bed and kept there until the urine is free of blood and albumen. This may be 6 to 12 weeks.

*Septic foci*—Septic tonsils, bad teeth, ear discharge, mastoid disease, septic spots on the skin and whitlows are all to be sought and thoroughly dealt with. As far as possible any surgical procedure should be left until the urine has cleared up, but treatment of a discharging ear or whitlow should proceed at once.

Teeth and tonsils can be left until the acute stages of the nephritis are over. There is naturally a fear that if the septic focus is left the kidney condition may recur and in the author's experience this does happen. Where there are masses of infected glands in the neck tending to keep up the sepsis and the temperature great patience must be exercised and it may be some weeks before it is safe to proceed with the teeth and tonsils. Recently Case (1939)<sup>1</sup> and others<sup>2</sup> in a series of cases stated that the removal of tonsils after a period of 9 to 12 months had no effect on the ultimate prognosis and that a similar series which was not tonsillectomized showed an equally good recovery rate.

The author thinks that this operation should be performed within the first two to six weeks or thereafter to be of any real help and it is certainly best done before the child is sent away to convalesce. When the tonsils are septic each fresh cold or sore throat produces an exacerbation of the nephritis.

**Diet**—During the acute stage drinks of barley water, fruit juice and water, sugar water or Imperial drink should be given in liberal quantities. The child should be offered his meal consisting largely of starch and milk food at regular meal times. For instance at breakfast, cereals, with raw or stewed fruit should be given. For dinner vegetable broth and milk pudding, and for tea thin bread and butter or bread and milk or some patent starchy food such as Benger's Food. It is difficult to overdo the fruit juice during this period. A high milk intake is not desirable and the quantity given should certainly not amount to more than a pint daily.

**Hæmaturia**.—When the urine shows only a moderate amount of blood in each specimen with a steady tendency for this to diminish in say a week or ten days nothing need be done. With large quantities, however, and where there is no tendency towards diminution the child may become

<sup>1</sup> Jones, The Ultimate Prognosis of Nephritis in Childhood. *Arch. Ped. Child.* No. 3 June 1939.

<sup>2</sup> W. W. Layne and H. S. Illingworth, Acute Nephritis in Childhood. *Quart. Jour. Med.* N.S. 32 Jan. 1940, p. 3.



progressively anæmic. Antistreptococcal serum, or horse serum 20 c. cm. given subcutaneously, will be found a valuable hemostatic in some cases.

**Bowels**—Salines constitute the best treatment or alternatively cascara or jalap. Metallic purges, such as calomel or grey powder are best avoided.

## 2 Latent stage (second stage)

**Symptoms**—There are usually no symptoms. Occasionally there may be acute headaches, with some vomiting termed migraine, and at that time slight puffiness about the eyes. As a rule however the general health is excellent. In only 5 of 98 cases were there symptoms (Case 1939). A 12 hour "Addis count" will show an excess of formed elements in the urine.

**Examination of the urine** shows traces of albumin. The red cells are present in excess and also the casts.

**Course**—Cases in this stage may heal completely or may pass gradually in the course of say 20 years into chronic nephritis and uræmia. Rarely they pass through the subacute or third stage.

TABLE XVI

CELLULAR ELEMENTS AND PROTEIN IN 12 HOURS NIGHT URINE IN NORMAL CHILDREN (SNOOK)<sup>1</sup> ADDIS COUNT

	Range	Mean Value	Standard Deviation	
Casts	0-29 000	1230	3 900	95% under 9 000 all were hyaline or granular
RBC	0-500 000	80 000	116 300	Above 600 000 suspicious
Protein	5-90 mgm	25.5 mgm	13.2 mgm	95% under 55 mgm 55 mgm suggests upper limit of normal

**Addis count**—To ascertain whether an old nephritic is in the latent stage or not, the following procedure is necessary. Drinks are restricted during the day time. The evening specimen of urine is voided and discarded. The urine is then collected during the following 12 hours—that is any urine passed during the night and the early morning, specimens are kept, and counts are done on the red blood-cells and casts and the amount of albumin is estimated. The normal range of these elements is seen in the table.

W. W. Payne states that the normal number of white blood corpuscles found in the urine, excreted over a period of 12 hours, is up to 1 million, and anything above 2½ million should be considered pathological.

## 3 Subacute nephritis (third stage)

This, formerly called 'parenchymatous nephritis' is merely a later stage of nephritis. It is rare. Usually there is an interval of one to three years between the acute and subacute stages. In two thirds of the cases there is no history of an acute stage at all this phase having passed unnoticed.

**Symptoms**—This disease is usually ushered in by the gradual onset of massive oedema, and vomiting, if there is nitrogen retention.

<sup>1</sup> A. W. Snook. Prognosis of Glomerular Nephritis in Childhood. *Amer Jour Dis Child* June 1939 vol 13 3

At intervals there may be an acute exacerbation of this subacute condition. At these times blood appears in the urine which is much diminished in quantity and both the blood urea and blood pressure rise rapidly. The oedema involves the face, limbs and all dependent parts including the scrotum penis and labia. The eyes may be completely closed. The heart is rapid and death may take place from dilatation and acute heart failure.

**Urine**—The characteristic feature is the large amount of albumin, and in many cases it will hold solid in the test tube. Red blood corpuscles may be present in excess and some granular and hyaline casts. The quantity is small but with any acute exacerbation it becomes smoky and still further lessened in quantity.

**Blood cholesterol** (normal average 160 mgm) is raised from 200 to 300 mgm per cent.

**Plasma proteins** (normal 6.9 gm) are lowered to 4.2 gm.

**Albumin globulin ratio** (normal 2 to 1) may rise to 1 to 1 or higher.

**Blood urea** is somewhat raised (normal is 20-40 mgm per cent).

**Blood pressure** is normal or somewhat raised.

**Pathology**—The kidney is swollen and engorged. Microscopically the tubules show cloudy swelling and fatty degeneration, and the epithelium tends to be shed into the lumen forming casts. The Malpighian tufts show marked destruction. Early interstitial changes can be seen. It is the picture of the large white kidney.

**Differential diagnosis**—The diagnosis from acute nephritis is made by the absence of a history of pre-



Fig. 36. Subacute nephritis, third stage. Note oedema of the lower extremities and face.

vious acute infections and by the gradual onset of the oedema. The urine with its high albumin content and relatively low red cell content is not that of acute nephritis. The diagnosis from nephrosis is made by the fact that in nephrosis there is no excess of red blood-cells or granular casts and no raised blood urea or blood pressure. Also there is no preceding acute nephritis.

**Prognosis**.—The disease is often fatal at this stage. Such patients have very poor immunity to pneumococcal and streptococcal infections such as pneumococcal peritonitis and erysipelas. Uremia may develop. Patients who survive tend to pass into the chronic stage from which they do not as a rule recover. Occasionally however a case heals.

**Treatment**. *General management*—The patient should be placed between blankets to encourage sweating. The greatest care should be

taken to avoid chills and woollen or flannel night-clothes are recommended

**Septic foci**—If obvious foci are present the outlook otherwise is so bad that the very considerable risk of operation is warranted. The author's experience is that on the whole, these cases do very badly indeed after operation. There is a tendency to become much worse with each passing cold or infection. The greatest possible care should be taken to protect against naso-pharyngeal catarrhs.

**Diet**—During an acute exacerbation a diet of milk and fruit is advisable but once this phase is over starches should be added. A salt free diet will help to reduce the oedema, but should not be persisted in if there is vomiting.

**Epstein's high protein diet** should be aimed at as soon as a good phase has been entered. This is a high protein moderate fat and carbohydrate diet and it includes both red and white meat. It should be introduced slowly. Oedema is an indication rather than a contra-indication for Epstein's diet. An occasional blood urea test will show whether the high protein diet is being tolerated or not. Should the blood urea be rising a reversion to low protein diet is indicated.

**Drugs**—Saljrgan half a cubic centimetre intramuscularly will produce a diuresis when required, and is recommended in some cases. The effect is of course, quite temporary. It is best to give from five to ten grains of ammonium chloride by the mouth for the previous twenty-four hours and again on the day of injection.

Urea (5 grains t d s) and Diuretin (8 grains t d s) may be given by the mouth and will help to produce a diuresis.

Thyroid has had no effect in the author's hands.

**Purges**—In order to get rid of the fluid accumulated drastic purges are necessary, and the stools should be watery rather than formed, but not so frequent that the child is thinned. Purges such as powdered jalap 10 to 20 grains or liquid extract of cascara sagrada a teaspoonful at bed time, or powdered rhubarb 5 to 10 grains or syrup of senna a teaspoonful at bedtime, are all suitable but it is best to avoid metallic purges such as calomel or grey powder.

**Tones**—Iron in the form of *syr ferri phos co* (Parnish's Chemical Food), with copper and manganese (see p. 178) half a teaspoonful three times a day and *ferri carb sacch* (sugar of iron) as much as will lie on a sixpence three times daily are advisable to build up the child. Extract of malt with cod liver oil and iron should be given during the winter months.

**Real or artificial sunlight baths** should be given care being taken to prevent the child from catching cold.

**Transfusion with serum**—Recently Aldrich and others have used four times concentrated serum intravenously to produce diuresis in cases of nephrosis with variable results. This treatment, with vacuum-dried

<sup>1</sup> E. W. Floerke and S. M. H. *Jour. Immunology* 1935, xlix 349. Aldrich, et al. *Jour. Amer. Med. Assoc.* 1934 (1) 179. C. A. Aldrich and H. H. Boyle. Concentrated Human Blood serum as a Diuretic in Nephrosis, *Jour. Amer. Med. Assoc.* March 1940 cxlv 1062.

human serum, appears to be a rational way of raising the serum protein of the blood

Edebohl's operation for decapsulation of the kidney has been practised over a number of years with occasional successes

**Drainage**—The introduction of Sonthey's tubes into the abdominal cavity legs and abdominal wall will be found useful but very risky. In this way many pints of fluid may be drained away. With such drainage a diuresis is sometimes started and the whole of the fluid from the tissues may quickly disappear. The reasons for this and its mode of production are not known but it has been proved on many occasions. Great care must be taken that no infection is carried in by these tubes, as patients are very likely to develop erysipelas at the site of puncture, or peritonitis when the abdominal cavity is entered.

**Hot air baths**—Sweating can often be induced by hot air baths, hot bottles, warm blankets and hot bags. Electric bulbs placed beneath the blankets act efficiently as also does hot air laid on from a special oven.

**Summary of treatment**—It is doubtful whether apart from good nursing, warmth, rest in bed and careful dieting together with suitable iron and vitamin tonics any treatment radically influences the course of this disease. Spontaneous cures occasionally occur after measles and, apparently for no reason at all, in a proportion of cases.

#### 4 Chronic nephritis (fourth stage)

In this stage there may be no history of a preceding acute attack of nephritis. It is comparatively rare in childhood, being made up of (a) a few latent cases and (b) those cases of subacute nephritis who have survived and passed into the chronic stage. Chronic nephritis is more often seen in adolescence or adult life.

**Symptoms**—These are those of irreparable renal insufficiency—headache, vomiting, retinal changes, hypertension, dry skin. The urine shows an increase of albumin, blood, epithelial cells and casts. On the other hand there may be dwarfism and symptoms of interstitial nephritis such as polyuria and increased thirst.

Such cases frequently present themselves with uræmia. The blood pressure may be up to 200 mm. of mercury (systolic), the blood urea rises to 200 or 300 mgm. per cent. Heart failure follows, but patients may live for many months or up to three or four years (Snook). With heart failure there may be some return of the œdema.

**Pathology**—All the elements of the kidney show destruction and fibrosis, and there is an increase of interstitial tissue, which tends to contract and produce the small granular or fibrotic kidney.

**Treatment**—A low protein diet with a low urea content, is indicated. Much bland fluid with fruit juice and starches, make up the caloric intake.

**Prognosis**—The outlook in this stage of the disease is practically hopeless. The author has never seen a case recover.

TABLE XVII

## NEPHRITIS

Features	ACUTE	LATENT	SUBACUTE DEGENERATIVE
Condition of patient	Nothing characteristic well or ill	Very well	Usually little but gross oedema
History of preceding acute infection	Usually present	Symptoms as	May be history of a preceding cold. Very rarely history of immediate preceding acute infection, such as acute tonsillitis or otitis
Oedema	None or moderate	None	Usually very gross
Albuminuria	Moderate	Usually very slight	Massive
Blood	Nearly always colours urine. Always large numbers of red cells	Excess of red cells	Very few red cells may be none for periods of several weeks
Casts (granular) (cellular)	Nearly always present often in large numbers	Excess	Usually absent or very few
Blood urea	Usually raised in first few days rapidly falling to normal	Normal	Normal or raised. If raised it usually remains high for a long time with remissions and exacerbations
Blood pressure	May be raised in first few days, rapidly falling to normal	Normal or rising	Normal or raised. If raised it usually remains high for a long time
Oliguria	Fairly frequent	None	May be present
Course	Rapidly resolves. Outcome as in diagram (p. 208)	Continues with eventual outcome as in diagram	Very chronic. Usually passes on into irreversible terminal stage (chronic nephritis). May pass into latent stage

TUMOURS OF THE KIDNEY<sup>1</sup>

In a 26 year period at Great Ormond Street Hospital Stern and Newns (1939) found *three true renal sarcomata, and one renal carcinoma* and one unidentifiable tumour of the kidney. They found, however, 26 Wilms' embryomata. In the same period they were able to report 25 tumours of the sympathetic nervous system, starting from the suprarenal. These will be dealt with elsewhere (see p. 373).

**Wilms' embryoma.** Definition.—A malignant tumour arising in close proximity to the kidney, but encapsulated from it, and containing a variety of embryonic tissue—immature glomeruli, and fully formed tubules, as well as muscle fibres and cartilage cells.

**Sex.**—Commoner in males than females.

<sup>1</sup> R. D. Stern and G. H. Newns, "Wilms' Embryoma," *Arch. Dis. Child.*, Sept. 1938, xiv, 192.

**Age.**—20 per cent. below 5 years, and 60 per cent. below 3 years.

**Site.**—Commencing in close proximity to the kidney. Commoner on the left side than the right.

**Symptoms.**—These usually commence insidiously: enlarged abdomen, abdominal pain, haematuria (uncommon) and wasting, with pallor and debility, are the commonest, in that order. On palpation, a huge firm and painless mass can be felt in one loin, and bowel can be felt over it at times. The blood shows nothing but anaemia, and there is no characteristic fever.

**Prognosis and treatment.**—Of 16 patients (Stern and News) who had nephrectomy, four were still alive 11 years, 8½ years, 4½ years, and 8 months after operation. Pre- and post operative deep X ray treatment improves the prognosis. The tumour is encapsulated and easily removed, recurrence is common in the operation bed. Metastases occur in the liver and lungs in 20 per cent. of cases.

**Diagnosis.**—The tumour may be mistaken for a hydronephrosis, but a pyelogram would exclude this. In a suprarenal sympathoblastoma the growth is more central, and secondaries in the liver and bones, especially the skull, are the rule.

### UREMIA

Uremia occurs when renal failure is so marked that waste nitrogenous products accumulate in the blood and tissues. At the same time, there is an accompanying fall in the blood calcium and alkali reserve. It may or may not be accompanied by a high blood pressure.

**Symptoms.**—Lassitude, muscular weakness, loss of appetite, coated tongue, and sore mouth, are some of the initial symptoms. Vomiting occurs, and sooner or later severe diarrhoea may be present. The temperature is, as a rule, subnormal, and coma appears at the end.

Symptoms of tetany, with carpopedal spasm, due to the low blood-calcium, frequently occur. Finally, the overbreathing or hyperpnea of acidosis, or the air hunger of alkalosis, is often seen as a terminal symptom.

**Prognosis.**—In nephritis which is reaching the terminal stage (stage 4), there may be a chronic state of uraemia with loss of appetite, nausea, and vomiting, and cramps in the limbs, and anaemia. In other cases, the symptoms may wax and wane, with periods of recovery between.

There are no retinal changes or fits in uraemia until the blood-pressure rises. The clinical diagnosis can be confirmed by a blood urea estimation.

**Cerebral symptoms due to high blood-pressure (hypertension).**—These are seen in acute nephritis (stage 1) or terminal nephritis (stage 4) where, as a rule, there is a high blood pressure with a failing kidney.

**Ætiology.**—The cerebral symptoms are said to be due to spasm of the cerebral vessels.

**Symptoms.**—Major epileptiform convulsions, often known as "hypertensive encephalopathy," occur, lesser degrees of these attacks are head-aches, giddiness, amaurosis and mono- or hemiplegia.

Retinitis is a constant feature, and flame-shaped hemorrhages are to be seen.

**Treatment.**—Lumbar puncture is indicated, and saline and glucose should be given intravenously, by the drip method. Sedatives, such as

chloral or amylal or rectal sedatives such as paraldehyde should be administered in suitable cases. Amputation is desirable if the blood pressure is very high and a low protein diet is indicated.

### NEPHROSIS

Nephrosis, a very rare condition has been confused with subacute nephritis (stage 3) or chronic nephritis (stage 4). Clinically the similarity is so great that some authorities do not agree that nephrosis is a separate entity.

**Symptoms**—The onset is insidious with gross edema and massive albuminuria.

**Urine**—No red blood cells are present but there are a few casts and a great deal of albumin.

**Blood**—The blood-cholesterol is raised but the blood urea and blood pressure are both normal. There is, therefore, no nitrogen retention.

**Ætiology and pathology**—The cause is unknown and the lesion appears to be a primary degeneration of the renal parenchyma with the main damage falling on the tubular epithelium.

**Prognosis**—In a true nephrosis red blood cells never appear in the urine, and after a varying period the edema and albuminuria clear up with complete recovery.

**Treatment**—No specific treatment is known but that suggested under subacute nephritis (stage 3) would be applicable. Particularly would this include four times concentrated serum intravenously and the administration of eurygan and ammonium chloride. There is a strong tendency for spontaneous cure in true nephrosis.

### RENAL DWARFISM

(RENAL RICKETS | RENAL INFANTILISM | CHRONIC INTERSTITIAL NEPHRITIS)

**Definition**—A rare condition of dwarfism in which marked rachitic changes develop with an upset in the calcium phosphorus blood ratio secondary to a gross renal lesion and azotemia (high blood urea).

**Ætiology and pathology**—This renal lesion may be congenital and be first noted when the infant is a few months old. The cause is not known but the kidney is seen to be extremely small not weighing more than perhaps one ounce. Microscopically it is grossly fibrotic and there are occasional cysts.

Renal dwarfism is to be more commonly seen in older children of 6 to 12 years brought to the orthopedic surgeon for knock knee. Occasionally a history of chronic pyuria may be obtained especially from the girls. Usually, however, there is no previous history and the Wassermann is negative. Undoubtedly some cases are secondary to pyelonephritis, and in such the disease passes into the terminal stage (stage 4) with symptoms of interstitial nephritis and bone changes. In such cases the thrust of the infection has fallen on the glomeruli and interstitial tissue.

The kidney is small and fibrotic and is composed largely of fibrous tissue. Occasional cases have followed on congenital cystic disease of the kidney. Valvular obstruction to the posterior urethra may give rise to this disease.

**Symptoms**—The infants are markedly rachitic. They fail to react to antirachitic treatment and vomit easily. A further examination will show polyuria, polydipsia and high blood urea (azotemia).

Older children present for knock knee. They are anemic with a tendency to develop bouts of uremia at intervals. The blood urea may be 100 mgm per cent. or more. There is often a cardiac murmur.

The urine is pale, excessive, with an extremely low specific gravity, say 1004. It contains a trace of albumin, some hyaline casts, and 1 or at most 2 per cent of urea.

There is usually excessive thirst, a high blood pressure, and hardened arteries, with retinal hæmorrhages appearing late.

The blood shows a low calcium, down to 5 mgm. per cent, instead of the normal 10 mgm., and a phosphorus of, say, 10 mgm. instead of the normal 5 mgm. Sunshine and vitamin D will not heal the rickets.

**Prognosis and treatment.**—The infants survive only a few months, perhaps a year or two at most. The older children rarely survive twenty years, and death takes place from uræmia. Calcium salts, such as calcium gluconate, 20 grains t.i.d., may be given on occasions in an effort to raise the blood calcium. The removal of the cause of the kidney lesion, such as a valve in the posterior urethra, should be undertaken if possible, but operations are badly tolerated.

A low protein diet and tonics should be given, and much care taken to prevent the contraction of infections.



## CHAPTER XII

### NERVOUS DISORDERS

#### LUMBAR AND CISTERNAL PUNCTURE

**Lumbar puncture** in children should be considered safe and simple if properly performed

**Dangers**—(a) *Sepsis*—This should be easily eliminated by carefully boiling the needle and by preparing the skin of the patient and hands of the physician

(b) *Impaction of the medulla and cerebellum* in the form of a cone in the foramen magnum by withdrawing fluid below when there is increased pressure above (usually due to a cerebellar tumour) Where cerebellar tumour is suspected and there is optic neuritis lumbar puncture should not be undertaken or should be done with extreme caution with a fine needle and very little fluid should be withdrawn

(c) *Breaking the needle*—A brittle needle should not be used Steel is best avoided nickel or platinum iridium is to be preferred

(d) *After effects*—In cases of meningitis and poliomyelitis or other neuro-pathological states nothing but good comes of the procedure Where the child proves to have a normal fluid the withdrawal of any considerable quantity may produce headache and vomiting This is corrected by keeping the child lying flat or raising the foot of the bed

**Indications for lumbar puncture**—Where the child has head retraction or neck rigidity and Kernig's sign together with other clinical symptoms of meningitis, lumbar puncture should be performed Papilloedema is a contra indication, as cerebral or cerebellar tumour should be suspected

**Pressure of fluid**—The normal pressure is 100 to 200 mm as shown on the manometer The pressure should be taken when possible but if the child is crying it is often very difficult In various forms of meningitis especially in the acute stage there is an increase of pressure This is also true of polio encephalitis and some brain tumours and abscesses A normal pressure does not exclude the last two Table XVIII gives the cerebro spinal fluid findings in various diseases

**Anæsthesia**—If the patient is mentally alert and normal it is best at all ages to give some general anæsthetic such as ethyl chloride gas or ether In some cases local anæsthesia such as 2 per cent novocain or freezing the skin with ethyl chloride may be selected

**Preparation**—The whole lumbar and sacral region is well indined especially over the crest of the ilium The patient is placed on the side with the knees drawn up and the head bent forward towards the knees so that the back is well curved The nurse or assistant grasps the patient behind the knees and at the nape of the neck, approximating the two hands In this way the spaces of the vertebra are drawn as far apart as possible allowing the needle to enter the spinal canal The left hand is

placed on the crest of the ilium, with the thumb opposite the space between the third and fourth lumbar vertebrae which is easily marked out. The needle is taken firmly in the right hand and the point pressed in between the spines of the vertebrae at this level in the mid line. On entering from half to three quarters of an inch a click is felt as the needle pierces the stretched spinal theca. The stylet is then withdrawn and the fluid flows freely. If the child is crying the fluid runs more freely than when he is quiescent, and if he is deeply anesthetized there is very little if any increased pressure. If the fluid is flowing too slowly the patient should be tilted so that the needle points slightly downward. If the needle is pressed forward through the spinal theca striking the anterior surface of the spinal canal a plexus of veins will be pierced so that there is a tendency for blood to appear in the fluid withdrawn. It is best therefore once the click has been felt to withdraw the stylet at once and not push forward.

How much fluid may be safely withdrawn?—As soon as the flow is seen to be very slow withdrawal should cease. In meningitis where there has been increased pressure it is well to run off as much as will flow freely, thus relieving pressure. Except in the extremely rare cases of cerebellar tumour all effects are unknown in children.

#### CISTERNAL PUNCTURE

**Preparation of the patient**—The back of the neck should be shaved well up to the external occipital protuberance and the skin well dried. The child is placed on its side and the head bent forward with the chin on the chest and firmly held there. It is well to give an anesthetic to patients who are quite conscious or normal mentally but in meningitis when the child is drowsy this may not be necessary.

**Technique**—With a well cleaned left hand the uppermost palpable spine of the neck should be felt and the lumbar puncture needle passed in upwards and forwards to the base of the skull. It should strike the skull behind the foramen magnum. The needle is now pressed up in the direction of an imaginary line passing through the centres of the ears. Should the base of the skull again be encountered the needle is withdrawn and pushed slightly farther forward. After possibly two or three attempts the needle, directed slightly farther forward each time, will pierce a strong ligament and enter the cistern. The penetration of this ligament is distinctly felt.

In the hands of the average physician this proceeding involves extremely little danger, and it is now accepted as a routine measure in certain cases in many teaching and children's hospitals. The advantages of cisternal puncture are that, when a puncture in the lumbar region has turned out to be dry, fluid is almost certain to be obtained here. Injected serum also, is much nearer the site of the lesion than when the lumbar route is used.

#### INTRACRANIAL ABSCESS

**Ætiology**—Intracranial abscess is usually a complication of ear disease. Rarely it may complicate frontal ectasia.

**Sites**.—Cerebellum (rare) and temporo-sphenoidal lobes (more common).

**Clinical picture**—After a chronic otitis media or mastoiditis, the temperature does not settle, the child does not improve and there is usually headache and vomiting. The physical signs depend on the site of the abscess and are similar to those of a tumour.

at that site. The pulse may be slow, with the increased intracranial pressure, and papilloedema usually develops.

**Cerebro-spinal fluid.**—This shows an increase of cells, usually lymphocytes but the chlorides are normal, and the fluid is sterile. Later, the cell count may rise and polymorphonuclear leucocytes are present. This suggests an extension of the infection to the meninges.

**Prognosis and treatment.**—Once the diagnosis has been made surgical interference should be undertaken. The prognosis is best in the temporo-sphenoidal group.

## INTRACRANIAL TUMOURS

**Cerebral tumours: incidence.**—The sexes are equally affected. In 102 cases reported from Great Ormond Street in a fifteen year period by Stern,<sup>1</sup> one half were under the age of five years and 30 cases occurred between the ages of two and four years. Fourteen out of sixty two proved cases occurred during the first two years of life.

**Situation.**—Tumours below the tentorium are twice as common as above the tentorium. Those below are situated in the cerebellum or pons and those above the tentorium lie in the fronto-parietal region, or at the base of the brain.

**Types of tumour.**—Two thirds of Stern's series were "gliomata." Highly malignant tumours were as common as more benign ones. Only one tuberculoma was found in this series. In order of frequency the tumours were—astrocytoma, medulloblastoma, glioblastoma, ependymoma, sarcoma and tuberculoma.

**Clinical picture.**—As in adults children show headache, vomiting and papilloedema with weakness or paralysis of various muscles. In cerebellar and pontine tumours the papilloedema may develop late in the disease. Since the skull of a child is soft and yields easily symptoms may be less well marked and develop at a later date than would be expected.

**Cerebellar and pontine tumours.**—These cases show headache, vomiting, and optic neuritis. Some mild hydrocephalus may be present with centrally placed tumours. Ataxia and hypotonia of the muscles especially of the limbs, nystagmus and ocular paralysis especially of the 4th and 6th nerves, are common. The gait is often staggering, and the head may be held to one side or other. The reflexes may be absent, normal or exaggerated and may vary from time to time. Paralysis of the facial muscles suggests that the pons or medulla is involved. Anaesthesia of the cornea is common.

**Hemispherical tumours.**—In many cases the first manifestation is fits. Weakness of one side of the body may follow and become increasingly more marked. Aphasia may be present. In more centrally placed tumours, ocular paralysis, especially of the third nerve, is common. In the later stages pyramidal tract pressure with rigidity develops.

**Pituitary tumours** are extremely rare. Visual defects, adiposity, and drowsiness are characteristic (Wylie).

**Pineal tumours** are also extremely rare. Signs of increased intracranial pressure, together with precocious puberty and paralysis of the upward movement of the eyes, are characteristic (Wylie).

**Cerebro-spinal fluid in tumours of the brain.**—Usually the cerebro-spinal fluid is normal, but the proteins and cells may be increased and the chlorides slightly lowered when there is rapid degeneration of the tumour.

Lumbar puncture should be performed with great caution when intracranial tumour is suspected.

X rays of the skull are valuable, as they may show calcification of a tumour or signs of increased intracranial pressure by widening of the sutures and "thumb markings" on the cranial bones.

Ventriculography and encephalography are useful means of investigating supratentorial tumours but should not be carried out where an infratentorial tumour is suspected. In ventriculography air is injected directly into the ventricles after removing some of the fluid. In encephalography air is injected through the lumbar puncture needle, after withdrawing fluid. From 50 to 100 c.c. of air are required to

<sup>1</sup> Ruby O. Stern, "Cerebral Tumours in Children—A Pathological Report," *Arch. Dis. Child.* Oct. 1917, xii, 291.

get a good photograph. X rays taken in appropriate positions will demonstrate tumours encroaching on the ventricles (space occupying tumours).

**Electro-encephalogram**—An investigation by this method may be of great use in localizing the tumour. For description, see p. 262.

**Treatment**.—Successful operation for cerebral tumour with complete removal may occur in a proportion of cases. Deep X rays or radium should be applied to tumours considered inoperable.

### INFANTILE HEMIPLEGIA

**Ætiology**.—Hemiplegia may be due to —

(1) *Birth injury*

(2) *Acute purulent encephalitis*—This is probably an infrequent cause, as the pathological lesion found in infantile hemiplegia is hemorrhagic and thrombotic, rather than inflammatory.

(3) *Hæmorrhage or thrombosis*—This is much the commonest cause. It may follow convulsions in whooping cough i.e. trauma or cerebral hæmorrhage may sometimes give rise to hemiplegia. It occasionally follows acute infectious diseases, such as measles, diphtheria and some unknown virus infections.

(4) *Epileptic convulsions*—In such cases the hemiplegia is transitory lasting a day or two.

(5) *Neoplasm*—Congenital hemiplegia may be due to malformation or a tumour growth of the brain.

**Age**—The condition may be present from birth (rare) may develop under the age of 3 years (common) it is rare after 6 years.

**Pathology**.—If examined at once when the symptoms appear a large hæmorrhage and one or more thrombosed vessels are usually found in the thrombotic cases. Such a case examined some years later would show sclerosis and scarring, with atrophy of the underlying brain tissue.

**Clinical picture**.—Usually the onset is sudden with a high fever, convulsions and loss of consciousness. After a varying period of a few hours or days, when the convulsions have ceased and the child returns to consciousness, the hemiplegia is discovered.

The child may recover completely, especially if the fit has been a simple epileptic one. If however hæmorrhage or thrombosis have occurred complete recovery is rare. The child is left as a rule, with a spastic leg and arm, and paralysis of the corresponding side of the face. The grasp is poor, and the arm is carried stiffly, with flexion at the wrist and extension of the fingers. The leg is stiff, and fails to grow as rapidly as the normal one and, since there is shortening of the tendo Achillis the child walks on the ball of the foot to compensate for the shortness. All the reflexes are increased on the affected side.

The mentality may not be affected but as a rule there is some deterioration in emotional control, or gross mental change may be present.

Convulsions commonly occur later, when scar formation or sclerosis has developed. Serious mental deterioration generally follows the convulsions.

**Treatment**—The child should be confined to bed during the active stage of the convulsions and onset. Lumbar puncture should be carried

**Mental condition**—The general impression is that all cases of spastic diplegia are gross mental defects but this is quite incorrect. A certain proportion, behind their mask of spasticity are bright and clever, and will make great progress with their lessons provided trouble is taken with them and they are able to get about unaided. The complication of convulsions however has a serious and deleterious effect on the mentality. Probably the majority of diplegics are backward mentally. This is in part due to their original injury and also to the life they lead, which prevents them from mixing with their fellows and getting about. As they get older they become shy and embarrassed, disliking strangers, and making the problem of their education still more difficult.

**Prognosis**—A few show very slight spastic paralysis and little or no mental deterioration. The vast majority however continue to show gross spasticity throughout life and are tremendously handicapped. In addition with cerebral impairment they are forced to live the life of invalids and most of them are institutionalized sooner or later. Only a small proportion are ultimately able to earn their living and become useful members of society.

**Treatment**—For the treatment of cerebral hemorrhage in the newborn see pp 68-69. Lumbar puncture and possibly blood by the intramuscular or intravenous routes and hypertonic rectal saline should be considered.<sup>1</sup> Hess states that the cerebro-spinal fluid of 90 per cent of all premature infants in the first fortnight of life shows xanthochromia.

**Treatment of convulsions**—A grain of chloral hydrate may have to be given hourly, or sufficiently often to control the convulsions in the newborn infant. When convulsions occur in older children lumbar puncture may be necessary or courses of one of the barbiturates such as luminal.

In older children the help of an orthopaedic surgeon will constantly be required. Tenotomies and special boots may be necessary. Contractures can be prevented by suitable splinting, massage, and manipulation.<sup>2</sup>

**Education**—Special educational facilities are bound to be necessary. Such children cannot attend the ordinary school and require much more individual attention. Schools for physically defective children cater particularly for them.

## EPIDEMIC ENCEPHALITIS

(ENCEPHALITIS LETHARGICA)

This condition popularly known as "sleepy sickness" may be defined as an acute inflammation of certain portions of the brain and brain stem giving rise to squint, stupor and various sequelae.

**Ætiology and pathology**—**Incidence**—This disease may occur from birth onwards. Of 25 cases analysed by the author the youngest was three months the oldest 11 years; the majority were more than three years of age.

**Sex incidence**—Males and females are equally affected.

**Seasonal incidence**—The disease is more prevalent during the winter months.

**The organism**<sup>3</sup>—It is appears to be a filter passing virus, which affects the basal

A. Monodet. Hyperton. Rectal saline for Intracranial Injury in the New born. *Brit Med Jour* June 16 1934 4, 106

<sup>1</sup> Donald Peterson. Evidence of Blood due to Hemorrhage into the Brain at Birth. *Jour Soc Massage and Med Gym* Sept 17 6 12 11

<sup>2</sup> *Advances in Pediatrics* Heinemann 1914 Vol. I p 47

ganglia and the nuclei of the third nerves, but any portion of the brain may be damaged and show a perivascular infiltration.

**Incubation period**—This varies from 7 to 14 days.

**Clinical picture**—The onset is manifested by fever, drowsiness, headache, double vision and vomiting. The child is comatose or drowsy, the speech is slurring and there is cataplexia. Ophthalmoplegia is one of the most characteristic symptoms.

**Cerebro spinal fluid**—The pressure is increased, also the albumin and the cells are raised from 10 or 15 up to 100 per c mm. The sugar is normal or increased and the chlorides are normal.

**Course**—The temperature settles in a few days or a few weeks and then characteristic changes may begin to appear. Diurnal somnolence and nocturnal wakefulness are frequent.

**Mental changes**—Some children become morally incorrigible, lying and thieving may result. There may be dullness or backwardness or even idiocy.



Fig 38.—Post-encephalitis lethargica. Note the obvious mental defect.

**Late sequelæ**—Parkinsonism, motor paralysis, myoclonus, respiratory symptoms with puffing attacks, Fröhlich's syndrome and epidemic hiccup, are seen.

**Differential diagnosis**—Encephalitis lethargica may be distinguished from—(1) tuberculous meningitis by the fact that it gives a negative tuberculin test and sputum test and normal cerebrospinal fluid chlorides. (2) from brain tumour and post diphtheritic paralysis which are excluded by the presence of a fever and absence of palatal paralysis. (3) from lymphocytic meningitis by the course of the disease and the small number of lymphocytes compared to those found in lymphocytic meningitis.

**Ultimate prognosis**—The outlook for life is good but in at least 75 per cent there is some degree of permanent cerebral damage. Parkinsonism and mental deterioration run a progressive course.

**Treatment**. (1) *For the stage*—Rest in bed is essential. Lumbar puncture is indicated

1. F. J. Hatten and G. J. Still, "Epidemic Stupor," *Lancet* 1918 I, 68. Donald Ross and A. J. J. Spencer, "The After-effects of Epidemic Encephalitis in Children," *Lancet* 1921 II 421. H. Morley Fletcher and J. D. Hollister, *Brit. Journ. Child Dis.*, 1931 xviii, 69. Cyril Burt, "The Young Delinquent," University of London Press, 1930. Mary M. Stevenson, *Arch. Dis. Child.* 1930 Dec. 11 p. 5.

careful nursing is demanded because of incontinence and the tendency to bed sores and oesophageal feeding may be necessary. Sedatives such as chloral bromide, paraldehyde, and aspirin may be called for and the diet should be milky, with starches and fruit juice.

(2) *Chronic stage*.—Severe cases require institutional treatment with special educational measures. For the treatment of Parkinsonism small doses of hyoscine grain  $\frac{1}{16}$ , tincture of belladonna minims 30 or tincture of stramonium minims 30 per day have been indicated.

Other forms of encephalitis.—Post measles and post vaccinal encephalitis are known to occur in childhood. (See pp 312-329.)

## MENINGITIS

### TUBERCULOUS MENINGITIS

**Ætiology and pathology.**—The disease is usually secondary to an old primary focus.<sup>1</sup> The initial lesion is usually in the bronchial lymph glands or mesenteric glands. In only one of 70 autopsies was the primary focus not easily recognizable. In 67 per cent the bronchial glands were caseous, but 58 per cent had caseous mesenteric glands in addition. The organism is usually the human tubercle bacillus and there is practically always a history of contact with some tuberculous individual. Only occasionally is milk the source of infection.

*Post mortem* the brain is seen to be congested and the coverings are studded with fine milary tubercles, particularly along the course of the vessels. In quite a high proportion of cases a tuberculoma is found in its substance. It is probable that such tuberculomata tend to soften and discharge into the subarachnoid space where the organisms are spread by the cerebro-spinal fluid. A varying degree of internal hydrocephalus is usual and this is probably the cause of death. (Plate II facing p 256.)

**Age incidence.**—The author's youngest case was a child of three months. The usual and maximum incidence for the disease is between one and two years, from which time the incidence rapidly falls to seven, after which it is uncommon. (Fig 62 p 339.)

**Seasonal incidence.**—Tuberculous meningitis is most common in the first quarter of the year when sunshine is almost absent, confinement indoors is at its maximum and fresh foods containing vitamins are most difficult to obtain. Infections are at their worst in this quarter and a primary tuberculous infection tends to become milary.

**Symptoms and diagnosis.**—The clinical picture is not always clear-cut as milary tuberculosis in some other part of the body may overshadow the meningeal infection. The pulmonary or abdominal symptoms may therefore predominate at first.

**Onset.**—In a clear cut case the onset is usually insidious. The child who was formerly active wants to be nursed or carried about, and there may be lassitude, headache, drowsiness and irritability. This is usually known as the *stage of irritability*. Constipation is usually present and all the early symptoms are often attributed by the parents to this fact. An

<sup>1</sup>Toussaint and MacIntyre. Sources of Infection in Primary Tuberculosis of Childhood. *Brit Jour Tuberc*, July 1936 xxx 195.  
 110 and 1200000. Tuberculous Meningitis—Is it a Preventable Disease? *Practitioner*, June 1936 cx 431.

occasional vomit, not necessarily in relation to food may be noted. Hyperæsthesia is prominent during this stage, and manifests itself in a complaint of pains and tenderness wherever the child is palpated. This lasts about one week.

The stage of compression follows the irritable stage. The pulse becomes slower, the headache more intense and a high cephalic cry may be given at intervals. During this second week the child shows signs of rapid wasting. The abdomen is scaphoid. He lies curled up on his side, dislikes a light, and may pull the bedclothes over his head. There may be slight stiffness of the neck muscles, but no definite head retraction.

The reflexes may be exaggerated or diminished and Kernig's sign is of little value for it is often absent.

The child may appear to frown or knit the brows probably because of the headache. If he is roused he answers a question sensibly but almost before he has finished drops off to sleep again. This stage also lasts approximately one week.

In the stage of coma there is a tendency for the temperature to rise and the pulse to quicken. Tache cerebrale is more definite. Flushing and sweating occur, and fine tremors of the hands are noticeable. The pupils become dilated, and may be unequal. The breathing is intermittent (Cheyne Stokes). The limbs are spastic and the child may have a series of convulsions. A squint is common. Swallowing ceases. The temperature rises to  $106^{\circ}$  or  $105^{\circ}$ , and death follows.

**Length of illness.**—This depends on the age and nutrition of the child, the average time being about 21 days. Too much stress should not be laid on any one symptom in the diagnosis, but the general picture should be considered.

**Mantoux and tuberculin patch test.**—These are usually positive, particularly when the solution for the Mantoux is a strong one say one in a hundred. Occasionally, however, the tuberculin reaction is negative, especially late in the disease.

**Sedimentation rate.**—In the early stages this may be much raised but in the late stage, just before death it will be normal.

**Differential diagnosis.**—The differential diagnosis depends upon the following considerations:—

(1) A constipated child may be irritable and out of sorts for a day or two, but this rights itself. The child with tuberculous meningitis goes steadily and slowly downhill from the start, with no real remission. A lumbar puncture will always differentiate.

(2) Bilious or infective acidosis attacks are differentiated by the profuse vomiting as distinct from the very occasional vomit of tuberculous meningitis.

(3) Virus infections such as polio encephalitis or encephalitis lethargica do not show a reduced chloride content of the cerebro-spinal fluid.

(4) Brain tumours have a slower and more insidious onset even than tuberculous meningitis.

(5) Uramic convulsions and psychosis are differentiated by the urine.



(6) Apical pneumonia may show some head retraction but the cerebro-spinal fluid is normal, and the chest X ray and the tuberculin test are unlike those of tuberculous meningitis

(7) Typhoid fever has a normal cerebro-spinal fluid, but gives a positive agglutination test, and there is looseness of the stools, rather than constipation, as a rule

**Cerebro-spinal fluid.**—This is clear, and under a varying degree of increased pressure. As a rule a cobweb-like clot forms on standing, and the fluid contains from 15 to 200 cells per c mm. which are chiefly lymphocytes. The sugar is reduced from the normal 55 mgm. per cent. down to 15 or 20 mgm. per cent. and the albumin is increased from the normal 25 mgm. per cent. to 40 or even 100 mgm. per cent. The chlorides are reduced from the normal 725 mgm. per cent. down as low as 500 mgm. per cent.

**Prognosis.**—In all cases of definite tuberculous meningitis, the outlook should be considered hopeless. A few cases have shown temporary improvement but relapsed later.

**Treatment.**—There is no treatment, apart from that of symptoms. Lumbar puncture relieves headache.

**Sedatives**, such as chloral, bromides, paraldehyde, amytal, luminal and avertin are all of some value, particularly in the late stages. Chloroform may be necessary to relieve the convulsions.

**Nursing**—The greatest care will be required to prevent a sore mouth, eyes and pressure points. Frequent attention is essential.

**Prevention**—Children who develop their primary tuberculous infection in the first two years of life run a distinct risk of primary tuberculous meningitis. The greatest care should be taken to prevent contact infection at all times but particularly in the first two years. The frequent use of the tuberculin patch test so that the primary infection is diagnosed at the earliest moment, will be most helpful, and prolonged convalescence with tonics and fresh air will heal the primary infection.

## CEREBRO-SPINAL MENINGITIS

(MENINGOCOCCAL OR POST-BASIC MENINGITIS—SPOTTED FEVER)

**Ætiology and pathology.** **Organism**—This is the meningococcus, a Gram negative diplococcus, which is as a rule intracellular.

The organism is thought to gain entrance to the blood stream through the naso-pharynx and during the first few days to be present as a septicaemia. Recent work suggests that the choroid plexus and ependyma bear the brunt of the cerebral infection, and the ventricles tend to fill with pus. The infection tends to be most severe at the base of the brain shutting off the intercommunicating foramina. Internal hydrocephalus tends to develop as in tuberculous meningitis.

In a severe case the whole surface of the brain is a thick felted mass of pus.

**Age incidence**—It is commonest in the first five years of life with a maximum at one year.

**Clinical picture.** Onset—There is high fever vomiting, and some looseness of the bowels in small infants; intense headache in older children.

**Fulminating type**—In some older children, almost from the onset, there is a purpuric rash and much toxæmia with a massive infection. This may prove fatal in 24 hours.

**Mild infection in older children**—The temperature is high, the headache intense, the neck stiff, and there is definite head retraction. Occasionally, this may be extreme. Kernig's sign is usually present. There is no photophobia, as in tuberculous meningitis. In fact, the child

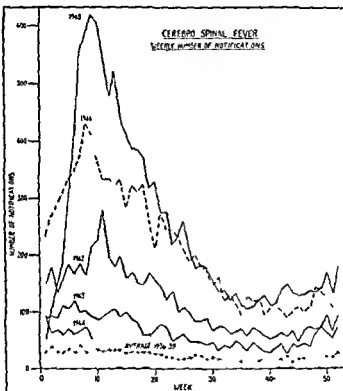


Fig. 39—Weekly notifications of cerebro-spinal fever in England and Wales  
(Reproduced by permission from the British Medical Journal)

tends to have wide-open staring eyes. He is conscious, and is able to talk, he vomits. In some older children the symptoms may be extremely mild with headache stiff neck and temperature only. These may subside and recur.

**Post-basic type.**—This is the type commonly seen in small infants. After loose stools there is a rise of temperature and marked head retraction. The fontanelle bulges, and, if the case is untreated, hydrocephalus develops.

**Diagnosis.**—An examination of the cerebro-spinal fluid is absolutely essential, it shows increased pressure. It is opaque, and contains many

Among the streptococcal cases, the hemolytic variety was implicated in 96 per cent

**Site of primary focus**—This was found in the ear or nose in 70 per cent of the cases in which the cause could be traced. Of cases secondary to diseases of the ear, nose and throat, the following frequency of infection was noted —

Auditory apparatus	90
Paranasal sinuses	18
Cavernous "	8
Post operative	8
Miscellaneous	3
Total	127

**Pathology.** (1) *Hæmatogenous route*—Various veins connect the meninges with the middle ear, nose, and paranasal cavities and infection may pass readily. Infected emboli may carry the infection to the meninges in some chest infections.

(2) *Osseous route*—Both from the ear and the nose (including the paranasal sinuses) infection may spread direct to the meninges. The layer of bone and dura separating these cavities from the meninges is so thin that any infection tends to pass through. As a rule, this type gives more warning and is slower in its onset than the hæmatogenous route.

(3) *Direct spread*—Particularly after trauma as a result of accidents and operations, there may be a direct infection of the meninges.

**Clinical picture.** *Onset*—This is sudden, and ushered in by head ache, vomiting, fever, and usually dislike of light with marked stiffness of the neck. The presence or absence of knee-jerks or plantar reflexes in a young child is not of great help. The temperature continues at 103° or 104° throughout the illness. The disease takes on extremely rapid course lasting as a rule not more than three days. By the second day small tremors and convulsions, with twitching of the eyes may be noticed. These convulsions later become constant and general. Kernig's and Brudzinski's signs may be positive, but in young children too much value should not be placed on their absence.

**Diagnosis.**—Apart from the clinical picture, the diagnosis is best made by examining the fluid after lumbar or cisternal puncture.

**Cerebro-spinal fluid**—This is increased in quantity the cells are increased to as many as 10 000 per c mm, all polymorphonuclears. The albumin is much increased. There is no sugar, and the chlorides are normal or slightly reduced. Organisms are grown readily.

**Prophylaxis.**—Wherever possible, septic processes in close relation to the meninges should be dealt with surgically, and treated with great respect.

**Treatment.** *Sera*—Antistreptococcal and antipneumococcal serum directly into the spinal theca, or intravenously, intramuscularly and subcutaneously, all have their advocates. They do not seem effective.

**Blood transfusion**—Such treatment appears to be given for general effect rather than a specific action on the meningitis. Intravenous saline and glucose drip is indicated where the patient is taking too little nourishment.

**Chemotherapy**—Particularly large doses of the Sulpha drugs are indicated from the very start, and it is often advisable to double the initial dose or two to get the concentration of the drug in the cerebro-spinal fluid from the beginning. When the pneumococcus or the streptococcus is implicated, M & B 693 (sulphapyridine), M & B 760 (sulphathiazole), sulpha diazine or sulphamethazine should be used. (For dosage, see p 383)

**Lumbar puncture and cisternal puncture** are indicated to reduce the increased intracranial pressure. Washing through the meninges, and continuous spinal drainage, have been advocated. Lumbar punctures should be continued at least once per day until the fluid appears to be normal.

**Eradication of primary focus**—When a focus is obvious, and can be removed, e.g. mastoid disease, surgical intervention is indicated.

## INFLUENZAL MENINGITIS

(PFEIFFER MENINGITIS)

**Ætiology and pathology**.—This is the fifth commonest form, and is about half as common as streptococcal or pneumococcal meningitis.

**Age incidence**—Influenzal meningitis is most common in infancy and between the ages of 6 months and 2 years. Less common throughout childhood.

**Organism**—This is the Pfeiffer's bacillus which is pleomorphic and Gram negative. It produces an acute inflammation of the meninges and surface of the brain like other forms of purulent meningitis.

The organism gains entrance to the meninges, probably from the blood stream, and is, as a rule, secondary to an infection in the lungs, nasal sinuses, or ears.

**Clinical picture**—There is an acute onset, with high fever, vomiting, rigor and a tendency to spasm or to convulsions. In every respect it resembles the onset of septic meningitis. Head retraction is seldom a very pronounced symptom. In infants the fontanelle bulges.

**Prognosis**.—Until the advent of the Sulpha drugs the mortality was more than 98 per cent. Recently this has been much reduced.

If untreated, the disease runs its course in about ten days, the cerebro-spinal fluid becomes thicker, and death takes place as in acute septic meningitis.

**Treatment**.—Roche and Caughey<sup>1</sup> (1939) report two cases of complete recovery after M & B 693 treatment. (For dosage, see p 383). In addition they gave a blood transfusion to each of their cases. Lumbar puncture was undertaken on several occasions. The author has not had satisfactory results from the Sulpha drugs in this disease.

Alexander<sup>2</sup> treated these cases with rabbit anti serum (influenza type B) (Squalids). The technique was as follows—

In order to speed up urinary excretion an intravenous drip was set up and the infant given as much as 40 c.c. per kilo of body weight per day of

<sup>1</sup> K. H. Roche and J. E. Caughey. Influenzal Meningitis treated with M & B 693. *Lancet* Sept. 16th, 1939 ii, 825.

<sup>2</sup> H. P. Alexander. Treatment of Hemophilus Influenzae Infection and of meningitis, septic and purulent meningitis. *Am. Jour. Dis. Child.* Aug., 1942 lxxi, 169.

either Ringer's or normal saline solution. The serum was added to the drip and one of the Sulpha drugs was given at the same time. The dosage of rabbit serum was based on the dextrose content of the cerebrospinal fluid—the lower the sugar content the greater the antibody requirement.

Alexander's recoveries were 82% out of 66 cases all of whom were over 7 months of age.

### TETANUS

**Incidence and age**—Tetanus may occur in the neonatal period (see p. 66). It is actually rare in the British Isles but is still found in farming districts. Of 21 cases (Cole 1935) 10 were under 13 years of age and the youngest was 8.

**Symptoms**—After an initial injury such as a cut splinter abrasion or contusion of the limbs there is a quiescent period which may be as brief as a few hours but is generally from 7 to 14 days. The onset is usually gradual with stiffness of the limbs and jaw muscles (*trismus*). Stiffness of the neck may be seen and in severe cases the back is arched. The upper part of the body only may be affected and risus sardonicus is often present. This has been termed cerebral tetanus.

**Prognosis**—In Cole's cases all those with severe wounds died. The outlook, however, was good where there was a long incubation period and from 60 to 75 per cent. of such cases recovered.

**Treatment**<sup>1</sup>—Tetanus antitoxin should be administered at once when ever a wound is fouled by farm material or in a farming district. From 5 000 to 10 000 units would be a useful prophylactic dose depending on age. Once the disease has commenced from 100 000 to 200 000 units should be given using the intravenous intramuscular and subcutaneous routes.

The wound should be carefully cleansed.

Ipecuan in doses of 0.1 c cm per kilo body weight prevents exhaustion from spasm.

**Inoculation against tetanus**—It is apparently now possible to give 2 doses of 1 c cm of formal toxoid antitetanus antigen at an interval of 6 weeks. It is not known how long this immunity lasts. It is often combined with anti typhoid vaccine.

### SEROUS MENINGITIS (OTITIC MENINGITIS)<sup>2</sup>

**Ætiology and pathology**—During the course of an acute otitis or mastoid infection signs of meningitis may occur. Lumbar puncture shows an increase of cells, usually polymorphonuclear and also increased albumin with normal chlorides in the cerebrospinal fluid. The culture may be sterile or show an occasional organism.

In some cases the condition subsides without further interference but usually the source of infection must be dealt with promptly. Probably

<sup>1</sup> Inoculation against Tetanus, *Lancet* Aug., 1935 II 3\*. L. Cole. The Treatment of Tetanus, *Proc Med Soc. June 12 1935* I, 1191.

<sup>2</sup> Discussion on Meningitis of Otitic Origin, *Proc Roy Soc. Med.* Dec. 1931 XXVII, 3\*2.

in every case there is a localized area of meningitis, adjacent to the primary focus

**Clinical picture.**—The child has headache, fever, irritability and some stiffness of the neck, and presents the picture of an early meningitis. In infants, the whole condition may occasionally settle down and leave some degree of hydrocephalus afterwards, or frank meningitis may develop if the source of infection is not eradicated

**Treatment.**—Paracentesis or mastoidectomy may be indicated, or even plugging the lateral sinus, tying the internal jugular vein when that has become infected

**Sulpha drugs.**—At the commencement of the illness full doses should be given (See p 363)

### ACUTE BENIGN LYMPHOCYTIC MENINGITIS<sup>1</sup>

(ACUTE ASEPTIC MENINGITIS, LYMPHOCYTIC CHORO-MENINGITIS)

**Ætiology and pathology.**—The infection appears to be a virus which is particularly lethal to mice. The lesion is confined to the meninges and choroid plexus, and may be particularly marked in the basal region (Hughes, 1937)

**Symptoms.**—The disease usually starts as a cold, with headache and coryza, the temperature is from 101° to 103° and Kernig's sign and neck stiffness are usually present. As the disease progresses there may be cranial nerve palsies and paraplegia, with incontinence and coma. Clinically the picture closely resembles that of a tuberculous meningitis

**Cerebro-spinal fluid.**—The cells are increased to 50 to 2000 per cmm, and from 90 to 100 per cent of these are lymphocytes. The fluid is sterile. The sugar, chlorides and urea are normal but protein is raised to 10 or even 100 mgm per cent.

**Diagnosis.**—The disease must be differentiated from tuberculous meningitis, in which the chlorides are lowered, as is the sugar content of the cerebro spinal fluid, also organisms may be found on culture, or on staining and the patch or the Mantoux tuberculin test is positive. Tuberculous meningitis gets steadily worse clinically, while in lymphocytic meningitis the condition becomes stationary for a week or two and then the child slowly recovers. The whole illness lasts from 3 to 10 weeks

Tuberculous meningitis occurs usually between the ages of one and two years, where is lymphocytic meningitis occurs in older children, more commonly above the age of four years

**Prognosis and treatment.**—Repeated lumbar puncture is all that is required for complete recovery. Very careful nursing is necessary, particularly where the child shows incontinence, coma and some paralysis. Gavage feeding may be necessary. The diet is composed largely of milk, cereals and fruit juices during the acute stage, with eggs and white meat together with finely sieved vegetables added when convalescence begins

### ACUTE ANTERIOR POLIOMYELITIS

(INFANTILE PARALYSIS, HEINE MEDIN DISEASE, POLIOENCEPHALOMYELITIS)<sup>2</sup>

**Definition.**—A neurotropic virus infection of the anterior horn cells of the spinal cord giving rise to a flaccid paralysis of one or more muscles

**Age incidence.**—Two thirds of the cases occur between the ages of one and three years. It is uncommon before one and after three years. It is evenly distributed between the sexes

<sup>1</sup> Findlay, Attack on History, *Lancet* 1936 I 650. W. Hughes, "Acute Lymphocytic Meningitis," *Brit Med Jour* May 1937 I, 10, 2

<sup>2</sup> As to the Ætiology of Meningitis, *Lancet* Med Jour Aug 22, 1947 p 721. Sir A. S. McNulty, "Epidemic Poliomyelitis, Epidemiology, Causes and Prevention," *Brit Med Jour* July 11, 1936 II, 57. R. V. Smith, "Incidence on Poliomyelitis and Paralysis of Poliomyelitis in Schools," *Proc Roy Soc Med* Jan 1939 32:115-173

**Seasonal incidence.**—In the British Isles the disease is most common in the months between July and October. Sporadic cases do occur, however, at intervals throughout the year, and may at times assume the proportions of a minor epidemic.

**Ætiology.**—The disease is due to a filter-passing virus, which may gain entrance through the nose, by the olfactory nerve endings, then by the olfactory nerve, to the optic thalamus and, by the posterior horn, finally to the anterior horns of the cord. The virus is neurotropic. The more recent investigations of Toomey<sup>1</sup> and Bodian and Howe<sup>2</sup> (1941) go far to prove that the infection gains entrance via the alimentary tract, including

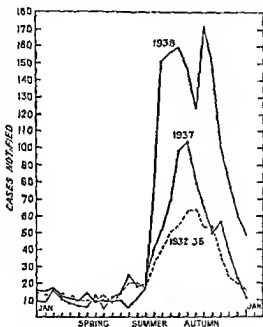


Fig. 40 Number of notifications of poliomyelitis and polio-encephalitis in fortnightly periods (Reproduced, by permission, from the *Lancet*.)

the pharynx. The virus has been isolated from the stools of patients for a period of some months and has been found in the sewage from hospitals nursing such patients. Healthy individuals may harbour the virus in their stools.

**Incubation period.**—Experimentally the incubation period varies from 8 to 12 days. Clinically it appears to be from 2 to 14 days, and as a rule less than 7 days.

It is difficult to state for what period a child should be considered infectious, particularly since the virus has been found in stools. It is generally agreed that a period of not less than 3 weeks is the minimum.

**Mode of spread of infection. Flies.**—The virus has been isolated

<sup>1</sup> J. A. Toomey, *Jour. Ped.*, 1941, xix, 303.

<sup>2</sup> D. Bodian and H. A. Howe, *Bull. Johns Hopk. Hosp.*, 1941, lxxv, 1, 2, and lxxix, 81.

from several species of fly including the house fly. It is not yet known whether the fly carries the virus externally or whether it is multiplying within the fly. Food may be contaminated by infected flies or the infection may gain entrance after an individual has been bitten.

**Stools**—Since the excreta of patients contain the virus, water supplies may be infected and in turn food may become contaminated.

It is clear that poliomyelitis should be nursed with the same precautions as any other infectious disease.

**Incidence of cases**—It is an extreme rarity to find more than one case in any one family but there are a number of instances on record of two three or more cases in one household. The natural incidence is very small as is shown in Fig. 40. In 12 000 public school children between 13 and 19 years of age over a period of 14 years 82 cases occurred (R. E. Smith 1939). An attack appears to immunize the individual against a second.

**Pathology**—The essential lesion is a destruction of the anterior horn cells of the spinal cord. The distribution may be widespread and affect the bulbar region or even the brain itself (polio-encephalitis). To the naked eye the brain appears congested and section of the cord often shows congestion or hæmorrhage. Microscopically there is cuffing of the vessels and the anterior horns may show invasion by lymphocytes and glial neuronophagic cells.

The cerebro-spinal fluid is clear with raised pressure and an increase of cells polymorphonuclears at first then lymphocytes. The protein is raised the sugar and chlorides are normal. The blood shows a polymorphonuclear leucocytosis.

**Muscles**—With disease rapid fatty degeneration and atrophy of the muscle-fibres takes place.

**Clinical picture**—The onset is sudden. The child may have what appears to be a sore throat or cold and the following day paralysis may occur. More often however there is a slower onset with fever, headache pains all over and some rigidity of the neck. In two to three days the temperature subsides and then paralysis is discovered. Fever is the rule and the limb pains may last as long as one month. The most constant symptom is neck rigidity so that the child objects to sitting up or flexing the chin on the chest. Pharyngitis is common. There is sometimes vomiting or diarrhoea. The muscular paralysis is usually at its maximum when first noted and tends to show improvement from this time on.

**Classification of the various types**—The lesion may be so widespread that clearly there must be a great variation in the clinical picture. The mental symptoms are more marked in cases of polio-encephalitis than where the cord alone is affected. When the motor cells of the cord are destroyed groups of muscles rather than individual muscles are picked out.

**Spinal form**—This is the most common type of paralysis. The legs are usually affected particularly the anterior tibial group of muscles. The arms are next most often involved but the diaphragm the intercostals the spinal muscles the shoulder girdle or the neck muscles may all show some weakness.



Any attempt to move the affected limb causes acute pain and this pain may continue for an indefinite period. If the cervical region of the cord is damaged, the arms, or the shoulder girdle, or the neck muscles may show paralysis, and the head is held up with difficulty. Where the thoracic segments are involved, paralysis of the abdominal and intercostal muscles occurs, in which event there may be bulging of the abdomen on the flaccid side. Involvement of the muscles supporting the spine may lead to curvature.

**Bulbar type**—This is a comparatively rare form which is commoner in older children. It has a relatively high mortality. It is commonly accompanied by mental confusion, and even hallucinations. Headache and giddiness are often present. Cranial nerve palsies occasionally causing squint or paralysis of the face muscles are characteristic. The commonest cranial nerves involved are those supplying the muscles of swallowing. Nasal voice is sometimes present. Choking attacks are a feature. This type occasionally follows tonsillectomy and adenoidectomy performed during the late summer and autumn.<sup>1</sup>

**Cerebral type (Acute encephalitis)**—A rare form accompanied by fever, vomiting, headache and convulsions and often showing hemiplegia. Mental defect or epileptic convulsions may result.

**Cerebellar type**—In this form well marked ataxia results but the patient usually recovers.

**Abortive type**—During an epidemic infected households or institutions present a number of cases which show the general prodromal symptoms but no paralysis. There may be temperature, sore throat and headache even stiffness of the neck, but these symptoms pass off in a day or two. They are probably mistakenly diagnosed as influenza. The blood serum, however, has been proved later to show antibodies to the virus.

**Diagnosis.**—(1) *Acute rheumatism* may give rise to painful limbs but there is no true paralysis, and the joints are often swollen and red. The neck is not stiff, and a cardiac lesion is frequent. Rheumatism commences usually at the age of 5 years.

(2) *Scurvy*—With blood in the urine, and swelling of the gums together with a history of lack of vitamin C, the diagnosis of scurvy should be easily differentiated. Scurvy occurs chiefly under one year of age the beading of the ribs is characteristic, and there are painful swellings on the thighs or elsewhere from subperiosteal hæmorrhage.

(3) *Meningitis* and obscure *encephalitis* must be differentiated by examination of the cerebro-spinal fluid. Increased cells and protein with normal chlorides and sugar, are characteristic of poliomyelitis.

(4) *Arthritis*—In this condition there is fixation of the joint, pain confined to the joint rather than the limb, normal cerebro-spinal fluid and little or no wasting of the muscles. Later, x-rays and the blood sedimentation rate will confirm the diagnosis of arthritis.

**Late diagnosis**—Within two or three weeks the affected limb becomes wasted, the muscles are soft when pressed between the finger and thumb, the power to make any given movement is lost, and there is a tendency for that limb to be colder than the other. Chilblains and trophic ulcers

occur. The reflexes are usually absent, and there may be no response to faradism.

**Prognosis**—The average death rate is about 10 per cent in an epidemic. The mortality is chiefly found in cases involving the respiratory muscles, the medulla and pons (bulbar type) or those with acute convulsion (polio-encephalitis).

Probably a very large number of abortive cases occur in each epidemic. After paralysis complete recovery is the exception, some degree of paralysis almost invariably remaining. The severity of the initial symptoms or degree of original paralysis is no guide to the ultimate extent of the lesion. When several muscles in one limb are affected there is a tendency for the bone on that side to cease its normal growth. The greatest amount of improvement occurs within the first six months after which the condition improves very slowly.

**Prophylaxis**—Isolation during the initial phase is usual and contacts should not return to school for a full three weeks. On the appearance of the second case in a school (Smith 1939) the school is best closed and the children sent home to be isolated there.

An attempt has been made to prevent the disease by instilling drops of zinc sulphate (1 per cent solution) into the nose. Tried in an epidemic (1937) in Canada, Alan Brown<sup>1</sup> reports that this treatment was a failure.

**Contacts**—From 20 to 30 c.c.m. of convalescent or failing this, adult serum injected subcutaneously appears to be a rational method of protecting contacts.

Vaccination against this disease has not been a success, and is not devoid of serious danger.

**Treatment**—*Acute stage*—The patient should be kept absolutely at rest. Any movement of the limbs or of the child, must of necessity pull on the inflamed spinal cord and nerves. Suitable splinting and a crig to carry the weight of the bedclothes are necessary. The acute pain felt in the affected limb may be much relieved and its duration shortened by the application of heat to the spine and affected part. Often this is best supplied by the careful application of fomentations or diathermy. Sedative drugs may be necessary to ensure sleep.

**Serum**—The injection of convalescent or adult serum subcutaneously, intravenously or intrathecally has various advocates. It is claimed that if this is given to children shortly after the onset of the paralysis the spread is limited. By others it is advocated in what appear to be the early stages. The proof of its value is inconclusive as, once the infection has occurred, the serum seems ineffectual.

**Mechanical respirators**<sup>2</sup>—In cases where the diaphragm and intercostals are involved a mechanical respirator is necessary. In some such cases after a few days the muscles recover but on the whole the mortality is extremely high.

<sup>1</sup> Canadian Pub. Health Jour. 1935, xxviii, 53.

<sup>2</sup> J. Macnamara and P. C. Morison. Polio-myelitis-encephalitis in Victoria (1935-37). Treatment by Human Immune Serum. *Lancet* 1937; i, 468. J. F. Leake, Polio-myelitis vaccine as being tried in L.S.S. *Jour. Amer. Med. Assoc.* Dec. 10, 1935, or 2167.

<sup>3</sup> G. Darlow. "The Use of the De L'Arrière Respirator in Polio-myelitis, *Newcastle Med. Jour.* 1935, xv, 1.

**Subacute and chronic stages** *Massage*—This should be commenced once the pain has passed from the limbs. The nutrition of the muscles is thereby improved. Passive movements and electrical treatment and more permanent splinting should be under the guidance of the orthopedic surgeon.

**Nursing**—To prevent trophic sores and chilblains suitable coverings for the affected limbs and the application of external heat will be required. A water bed or ring pillow and much attention to the pressure points by alcoholic rubs, are necessary.

## PROGRESSIVE SPINAL MUSCULAR ATROPHY

(WERNIG HOFFMANN PARALYSIS)<sup>1</sup>

This very rare condition is familial and independent of sex. It occurs chiefly in infants, the first symptoms being usually noticed shortly after birth though in some cases they do not appear for a few months.

**Clinical picture.**—A healthy infant is noticed to be unable to move its limbs.



Fig. 41.—Wernig-Hoffmann paralysis (progressive spinal muscular atrophy). Note the over action of the diaphragm causing protrusion of the abdomen with sucking in of the sternum. The arms hang in a lifeless position due to their almost complete paralysis.

properly at the shoulders and hips while the hands and ankles, elbows and wrists move freely and the grip is good. Later it is noticed that the diaphragm tends to overact, the abdomen being large and pendulous (Fig. 41) whereas the intercostals act feebly. The grip and movements become less and less good until the child is only able to move the toes slightly. The facial muscles and power to swallow are unimpaired. The neck muscles are weak so that the child is unable to hold up its head. Death takes place after some weeks or months from some complication such as pneumonia. The duration of the disease and age of onset vary greatly, some children living to the age of 2 or 3 years, the process being extremely chronic.

**Etiology and pathology**—The cause is unknown. Post mortem nothing is seen with the naked eye but microscopically the anterior horn cells show slight or gross destruction. They are few, small and have no dendrons. Ponglik speaking, the picture resembles that of a chronic anterior poliomyelitis, if such a conflict exists.

<sup>1</sup> J. G. Greenfield and Ruby O. Stern. "The Anatomical History of the Wernig-Hoffmann and the Oppenheim Forms of Infantile Muscular Atrophy." *Brain*, 1917, 40, 65. Donald Paterson. *Wernig-Hoffmann Paralysis*. 1929, 22, 43.

The muscles are everywhere deficient or absent the pectorals and flukes being mere thin sheets

**Treatment.**—There is no treatment

### AMYOTONIA CONGENITA

(OPTECHISM & DISEASE)

This rare condition has a pathology similar to that of progressive spinal muscular atrophy (Werdnig Hoffmann paralysis). The characteristic feature is that the child is born hypotonic flabby weak and unable to raise the head or make any movements and that there is a steady progress towards recovery, so that ultimately the child is able to get about and do moderately well. The treatment consists of massage electrical treatment, sun baths and general tonics and good feeding.

In view of the similarity of the pathology of amyotonia congenita and Werdnig Hoffman paralysis these two diseases must be one and the same despite the wide divergences in the clinical picture.

### PINK DISEASE

(ERYTHRODEMA POLYMPURITIS INFANTILE ACRODYNIA)

**History.**—The first description was by Selter,<sup>1</sup> of Solingen in 1903. Another description was given by Swift<sup>2</sup> of Australia in 1911.

**Early clinical picture and mode of onset.**—In the cases reported, now running into many hundreds the youngest is about 4 months, and the oldest 6 or 6 years. The vast majority, however, occur between the ages of 9 and 18 months.

**Sex.**—Both sexes are equally affected.

**Onset.**—In nearly every case the disease starts as a slight cold. During this time there is a temperature, up to 100° or 101°, and the child is "out of sorts." The cold improves, and there is an interval of about a fortnight, during which there seems to be a slight but definite improvement. From then on, however, there is a steady deterioration in the child's physical and mental condition.

**Mental upset.**—In perhaps no other disease is the child more miserable and nothing will comfort it. A state of complete negativism develops, everything is refused. There is loss of appetite, all the good habits and training fail and insomnia is most marked. The child with pink disease presents a picture of utter misery. The mind is not affected. He takes notice of his surroundings and understands but is markedly preoccupied with his own unhappiness.

There is usually insomnia, but the ordinary hypnotics have little or no effect. Wasting occurs since the refusal of food is obstinate.

**Rash.**—A rash appears on the trunk about a month after the initial symptoms. This is sudanese, and the hands and feet show a redness in the glove and sock distribution, and have the appearance of having been dipped into boiling water. When touched, however, they are cold and slightly swollen and do not get on pressure. In time these "sodden hands" and feet tend to peel and the condition waxes and wanes, sometimes better and sometimes worse. The rash on the trunk which is a typical sweat rash, also waxes and wanes and because of the constant moisture of the skin, furunculosis is apt to develop. The skin of the hands and feet

<sup>1</sup> P. Selter, *Arch. A. u. N. d. H. 1903* 22:471, 475.

<sup>2</sup> H. Swift: "Erythroderma," *Trans. Australasian Med. Cong.*, 1912 session (Melbourne 1911), 31.

irritates the child, so that they are constantly being rubbed one against the other. A much older child once told the writer that his hands and feet felt like fire.

**Attitude in bed**—A typical attitude except in very tiny infants is that the child lies on his face, with the knees drawn up and the face buried in the pillow, in what might be termed the kangaroo or knee-elbow position. The head is slowly rolled from side to side, in a burrowing motion into the pillow in an effort to allay the irritation of the forehead.



Fig. 42. Pink disease.

1. Look of extreme irritation on child's face.  
 2. Attitude of the child in bed.

cheeks and tip of the nose which look red from friction. If the child lies on his back the mouth hangs open in the "young gosling" position. This is due to the marked hypotonia of the masseter muscles.

**Photophobia**—This is rather variable and where it exists is one of the reasons for the attitude in bed. At times it is the first and most marked symptom and in some cases almost the *only* symptom of the disease. At some time or other, however, in most cases the child has well marked photophobia. The eyes are kept tightly shut and only opened a little

when the room is quite dark. This symptom does not last more than two or three weeks, as a rule.

**Muscular hypotonia**—In addition to the weak masseters, all the muscles are soft and flabby. The grip is poor. The knee jerks are very difficult to elicit, or absent. The buttocks are wasted, and the child does not want to move about. There is no definite paralysis. Rectal prolapse is not infrequent.

**Late clinical picture**—As time progresses widespread symptoms appear, which can be best explained as due to a sensory polyneuritis.

**Condition of the mouth**—Small ulcers appear on the tongue and in the cheeks. These add to the difficulty of getting the child to take his food. Some of the teeth become loose and may be swallowed and appear in the stools.

**Hair and nails**—The hair tends to fall out, and in severe cases large bald patches appear. The nails may fall off in more marked cases although this is very rare.

**Sensation to pin pricks**—There is very definite dulling of sensation to pin pricks over the feet, hands and tip of the nose. Nothing characteristic is found in the fundi or in the respiratory system.

**Temperature**—A slight temperature is usually present at the onset of the disease, but throughout the remainder of the illness there is little or no fever. There has certainly been none in any case seen by the author, beyond an occasional slight rise if the condition were complicated by a boil or naso-pharyngeal infection.

The cerebro-spinal fluid is normal and the urine is also normal, although in some cases albumin and even pyelitis have been present as a complication. The blood shows slight leucocytosis—15,000 to 40,000 white cells per c mm.

**Tachycardia**—A prolonged tachycardia is one of the most characteristic features. Both day and night over as long a period as four to nine months, the heart races at 120 or even 140 beats per minute. Blood pressure readings have been taken and are almost invariably increased. The clinical picture suggests that there is some disturbance of the vegetative nervous system as shown by the sweating, rapid pulse, raised blood pressure, vaso-motor paralysis at the extremities, salivation, rhinorrhoea, photophobia and trophic disturbances. The sedimentation rate is normal.

**Ætiology. Diet**—Some maintain that this is really a deficiency disease and that there is some dietetic error particularly in regard to vitamin B. A condition somewhat similar was produced in rats by feeding on a diet deficient in protein, egg white being the only protein given (Emdley and Stern<sup>1</sup>). The majority however see no reason to consider diet a factor. Breast fed infants develop the disease, as do children carefully brought up in families where the feeding is beyond reproach, with all vitamins offered in liquid amounts. Concentrated vitamins do not cure, and Petrus concentrated vitamin B complex failed to improve the condition.

**Poisoning with metals**—In France an epidemic of a somewhat similar disease known as *acrodynia* was described in adults, and was

<sup>1</sup> C. M. Emdley and E. O. Stern. A type of disease in the Rat resembling Infantile Disease in Man. *Arch. Dis. Child.*, 1929, 4, 1.

that the child cannot say "Bilbe Button" without betraying paralysis. A clinical examination reveals —

(1) *A local lesion at the site of the infection* — If the infection is nasal or pharyngeal the palate is involved. The infection may, however, have been in the vulva, penis, anus, umbilicus or ear, and may have caused paralysis of the surrounding nerves.

(2) *A specific lesion* — In all cases of diphtheritic neuritis eye accommodation is affected to a greater or lesser degree. For instance, when the child is reading he puts the book down at short intervals to rest, this is because his power of accommodation is impaired. It may be severe enough to cause squint or ptosis.

(3) *A general lesion* — This is a general affection of the nerves throughout the body, so that all the muscles are weak. The knee jerks are lost and the diaphragm or the intercostals may be affected, interfering seriously with respiration and the power to cough. The duration of the illness is from two to six weeks or longer.

**Differential diagnosis** — Post-diphtheritic paralysis must be distinguished from toxic polyneuritis, where palatal paresis is not a feature and the onset of paralysis is rapid. In the bulbar type of infantile paralysis there is fever while post diphtheritic palsy is afebrile.

**Complications** — When the diaphragm or the intercostals are seriously affected, the child is extremely liable to respiratory infections and to massive collapse of the lungs. Coupled with this, there is difficulty in swallowing due to palatal paresis, and there is a great tendency to aspiration pneumonia on this account. One of the most serious complications is vomiting, and it may be necessary to feed rectally for the time being with a solution containing 7 to 10 per cent. of glucose, or to give saline and glucose, 5 per cent., by intravenous drip. Feeding with a nasal tube or stomach tube small quantities at a time, is often useful for a short period.

Sudden death from suprarenal hemorrhage is recorded in a certain proportion of these cases. Cardio failure may occur in the acute stage of diphtheria, and also as one of the sequelae, and causes the sudden death of a few patients.

**Prognosis** — Gross post diphtheritic paralysis, especially when the diaphragm or intercostals are involved, is serious with mortality of 10 per cent. or more.

**Treatment. Antitoxin** — As a rule, when the diagnosis of post diphtheritic paralysis is made, some weeks have elapsed and in some cases antitoxin has already been given but in insufficient quantities. Where it has not been given, a swab should be taken of the nose and throat, and from 8 000 to 20 000 units of antitoxin may be administered, no very great benefit should be expected from this, as the damage has already been done to the peripheral nerves.

**General management** — The child must be nursed as flat as possible, thus throwing little work on the heart, but when the diaphragm or intercostals become involved and swallowing is difficult, the head should be placed in such a way that mucus does not tend to collect in the back of the throat. Constant swabbing with gauze on forceps, or a suction apparatus, helps. If the diaphragm or intercostals are seriously involved it may be necessary to use a Drinker or Vassiloff respirator.

**Diet** — This should be fluid or semi solid, depending on the child's capacity to swallow. Very often semi solids are managed best, milk, thickened with some starchy preparation such as groats or Benger's food, being most successful. Feeding may be by nasal tube in difficult cases but care must be taken.

**Drugs** — As a nerve stimulant, strychnine is most efficacious, and should be given hypodermically at four hourly intervals during the acute stage. The dose is from  $\frac{1}{100}$  to  $\frac{1}{50}$  of a grain for children between the ages of two and eight years. In the earlier stages, for children of this age, it may be exhibited best in the form of tincture of our vomica, 1 to 3 minims, three times daily.

Atropine is useful to dry up undue secretions, and to cut off the cardiac depressors. The dose for children between the ages of two and eight years is from  $\frac{1}{2}$  grain to  $\frac{1}{10}$  grain.

Cardiac stimulants, such as digitalis, are not indicated, but brandy or coramine may be administered during the acute stages. The essential in these cases is careful expert nursing, which is more important than treatment.

**After-care** — As the palatal paralysis clears up, the power of accommodation improves and the knee jerks return, the child should be allowed an extra pillow, and finally may sit up and get about. Massage to the limbs at this stage is valuable. It may take six or eight weeks, or even longer, before the paralysis has improved enough

for the child to get up. A careful watch should be kept for signs of heart block, and a cardiac irregularity may be ascertained by an electrocardiographic tracing. In the later stages, general tonics, sun baths, and fresh air are strongly indicated.

### FACIAL PARALYSIS

**Ætiology.**—The paralysis may be due to a congenital or an acquired defect in the nerve or its nucleus. Congenital defects are exceedingly rare.

**Acquired lesions.** (1) *Supra nuclear lesions*.—These may be caused by a tumour, abscess, hæmorrhage, or infection of the cortex or internal capsule. This is best seen in hemiplegia. Voluntary movements are more affected than emotional ones.

(2) *Nuclear lesions*, usually caused by poliomyelitis of the bulbar type, may be the result of a tumour in the ponto-cerebellar region. In this case they are usually accompanied by a sixth nerve paralysis.

(3) *Infra nuclear lesions*.—If the lesion is in the *pons* and is due to poly-encephalitis, it will be a crossed type of paralysis. If at the *base of the brain*, then a ponto-cerebellar tumour, meningitis, or even a fracture, may be the cause. If in the *temporal bone*, then otitis media or mastoiditis should be suspected. If in the *peripheral nerve*, the following are the possibilities:—(a) injury with forceps at birth, or trauma from an accident; more commonly (b) catching cold in a nerve, as in Bell's palsy; (c) diphtheria, toxic polyneuritis, arsenic or lead poisoning.

### BELL'S PALSY

**Pathology.**—The nerve has become inflamed, swollen and is compressed during its course through the bone.

**Onset**.—This is rapid when due to a 'cold' but much slower when due to otitis media or mastoiditis.

**Symptoms.**—The eye cannot be closed, the forehead cannot be wrinkled, and the lips are not separated on the affected side when the child is asked to show the teeth. The child cannot whistle, and smiling shows the side affected. Food tends to collect in the paralysed cheek, and the conjunctiva may become infected on that side.

**Course.**—Recovery is the rule when due to a 'cold'. If due to inflammation of the ear, recovery takes place after operation. Nuclear lesions may show no recovery, or only slight recovery.

**Treatment.**—Little or no treatment is required when due to a 'cold,' but massage and electrical stimulation are indicated when the acute stage is over. When due to otitis or mastoiditis, surgical intervention is necessary. If in six months there is no improvement, an open operation with nerve anastomosis is indicated.

### TOXIC POLYNEURITIS

This is a rare condition, probably infective.

**Symptoms.**—After a sore throat, or what appears to be "influenza" there is a variable interval of several days or weeks before weakness of the muscles occurs. The limb muscles are affected first, and these are tender when touched. The reflexes are lost, and almost complete loss of power is present, with foot and wrist drop. Facial and other cranial nerve palsies may occur.

The cerebro-spinal fluid is usually normal, but there may be an increase of protein and lymphocytes (Wyllie).

**Prognosis.**—Recovery is the rule.

**Pathology.**—There is a diffuse inflammation of the peripheral nerves.

**Treatment.**—Rest, massage, splinting to prevent deformities, electrical treatment, and careful nursing should be supplied. If the intercostals or diaphragm are involved, it may be necessary to use a Drinker or Nuffield respirator, and feed by stomach tube.

### THE MYOPATHIES (MUSCULAR DYSTROPHIES)

This group of diseases is characterized by progressive weakness of the muscles, and sometimes wasting or replacement by fat or fibrous tissue.

**Incidence.**—The myopathies are both familial and hereditary, the disease on the whole being passed down through the females to the males, but not necessarily all the members of a family are affected.



**Etiology**—Nothing is known of the causation. Any muscle or group of muscles may be involved. The commonest types are —

- (a) The pseudo hypertrophic muscular dystrophy, to which the great majority of cases belong
  - (b) The juvenile (Erb) type (scapulo humeral type)
  - (c) The facio scapulo humeral type (Landouzy Dejeune)
- Detailed description of one type only is given —

#### PSEUDO-HYPERTROPHIC MUSCULAR TYPE

**History and clinical picture**—Usually the subject is a boy who is seen at the age of 4 to 6 years because he is not able to walk upstairs or to get up from the floor in a normal manner. It appears that he was late in walking but soon learned to run



Fig. 43.—Case of pseudo hypertrophic muscular paralysis third stage of rising from the ground

about more or less as other children, then he gradually lost the power of mounting stairs or getting up from a reclining position. In rising from the floor he gets up by rolling on to his abdomen, pressing himself up on his elbows, getting on to his knees then gradually climbing up his own thighs into the upright position.

The calves of his legs are much larger and apparently better developed than normal but the shoulder, pectoral and scapular muscles are very deficient or almost absent. In attempting to lift such a child from the floor by hands under the arms he almost slips through the hands because of the deficiency of muscle in the shoulder girdle. The arms are raised above the head with difficulty.

**Walking**—He walks in a characteristic fashion with marked lordosis and an apparent difficulty in getting the feet to the ground. There is a shortening of the tendo Achillis and to balance himself the child must lean well back. He has a waddling gait. In going upstairs he pulls himself up by the banisters.

The knee jerks are present at first but ultimately disappear.

**Prognosis.**—The other myopathies have the same essential pathology although other muscles are involved. The outlook is uniformly bad as the disease tends to be progressive. Patients seldom survive to the twentieth year.

**Treatment.**—There is no specific treatment. Such children should be kept on the move as once they are taken off their legs for any reason they tend to show less ability to get about afterwards. Massage, active and passive movements, radiant heat and artificial sunlight should all be tried. Tonics and changes of air are also important. Tenotomy may be necessary. Organotherapy and injection of fibrolysin and other muscle preparations have not proved successful in the author's hands.

**Utinins.** F. Bicknell<sup>1</sup> has treated cases with fresh dried whole wheat germ  $\frac{1}{2}$  oz. twice daily and claims good results. A preparation of this is Ephynal (Roche Products) one 3 mgm. tablet three times daily. On the other hand W. Antopol and C. F. Schotland<sup>2</sup> claim success with a Vitamin B concentrate.

### GROSS MALFORMATION OF THE SPINAL CORD SPINA BIFIDA\*

Spina bifida is a failure of the laminae arches to unite and is most commonly found in the lumbar region but may be present at any point in the whole length of the spine. About 30 per cent. of these cases may expect a relatively normal life.

(1) **Spina bifida occulta.**—In this condition there is a defect in the laminae arches, with no protrusion of the meninges. Nothing whatever may be shown externally and the diagnosis can only be made by X-ray. Commonly however there is a dimple or tuft of hair over the region and a fibrous band may be present connecting the skin and the dura covering the meninges. Symptoms attributed to spina bifida occulta are persistent incontinence of urine and sometimes of feces. In all such cases it is well to have an X-ray of the spine to exclude this condition. Usually however spina bifida occulta gives rise to no symptoms.

(2) **Meningocele.**—This is the commonest form of deformity and the protruding sac of dura contains cerebro-spinal fluid only. The swelling may be of great size and present an alarming picture at birth as the bare meninges are moist and bulging. As a rule, it is best to surround the sac with sterile lint and keep these parts scrupulously clean. Sterile vaseline may be required in the early stages and, later, dusting powder. With patience the sac gradually contracts and in a few months in the vast majority of cases disappears without surgical treatment. Very often hydrocephalus will develop if surgical interference is attempted at this stage.

(3) **Meningo-myelocoele.**—The sac in this condition contains nerve endings as well as cerebro-spinal fluid and the nerve roots may be attached to the wall of the sac. Here again surgical treatment is not indicated. In such a condition there may be paralysis of the bladder and rectum and paralysis of the lower limbs with sensory changes. The outlook in such cases is not good.

### MENTAL DEFECT

This may be defined from a social standpoint as a state of incomplete mental development of such a kind and degree that the individual is incapable of adapting himself to the normal environment of his fellow in such a way as to maintain existence independently of supervision, control or external support. (Tredgold 1937)<sup>4</sup>

The legal definition (Mental Deficiency Act 1927) says mental defectiveness means a condition of arrested or incomplete development of mind existing before the age of 18 years whether arising from inherent causes or induced by disease or injury.

\* F. Bicknell, *Lancet* Jan. 6 1910, 1, 30.

<sup>1</sup> *Four Sister Med. Assoc.* March 23 1940, vol. 1958.

<sup>2</sup> Garrod, Batten & Threlkeld—*Diseases of Children*, Arnold, 4th Edn. p. 511.

<sup>4</sup> A. F. Tredgold—*Mental Deficiency*, Baillière Tindall & Cox 6th Edn. 1937.

F. D. Ingraham and H. Gwan, *New Eng. Jour. Med.* 1948, cviii, 5-7.

Four classes of persons who are mentally defective are recognized by this Act—idiots, imbeciles, feeble-minded persons and moral defectives. The definitions are—

**Idiots**, that is to say, persons in whose case there exists mental defectiveness of such a degree that they are unable to guard themselves against common physical dangers

**Imbeciles**, that is to say, persons in whose case there exists mental defectiveness which, though not amounting to idiocy yet renders them incapable of managing themselves or their affairs or, in the case of children of being taught to do so

TABLE XIX

CLASSIFICATION OF THE CLINICAL VARIETIES OF AMENTIA <sup>1</sup>			
Approx. per cent.	Clinical Variety	Pathogenesis	Form
30 50 05 05 10	1 SIMPLE 2 MONOCOLISM 3 MICROCEPHALIC 4 SCIEROTIC Miscellaneous rare types e.g., Nervous Hypertelorism Oxycephaly, etc.	A <i>Cerebral Group</i> Imperfect neuronic development due to defective germ potentiality (may be accompanied by gross lesions or special pathological processes)	Primary
50	5 TRAUMATIC	B <i>Traumatic Group</i> Arrest of neuronic development due to brain injury	
40 30 10 30	6 MENINGITIC 7 ENCEPHALITIC 8 HYDROCEPHALIC 9 SYPHILITIC	C <i>Infective Group</i> Arrest of neuronic development due to lesions of brain or membranes consequent on infection	
0.5 10	10 AMAUROTIC 11 EPILEPTIC	D <i>Degenerative Group</i> Arrest of neuronic development as an incidental phase in a degenerative process (which may be due to germ abnormality)	Secondary
0.5 0.3 0.2 1.5	12 CRETINISM 13 NUTRITIONAL 14 ISOLATION Miscellaneous rare types e.g. Schilder's Wilson's Gaucher's etc.	E <i>Deprivative Group</i> Arrest or imperfection of neuronic development due to deprivation of hormones nutritive or sensory stimuli	
100.0			

**Feeble minded persons**, that is to say, persons in whose case there exists mental defectiveness which though not amounting to imbecility, is yet so pronounced that they require care supervision and control

<sup>1</sup> Taken from Tredgold's "Text-book of Mental Deficiency" by kind permission

for their own protection or for the protection of others, or, in the case of children, that they appear to be permanently incapable by reason of such defectiveness of receiving proper benefit from instruction in ordinary schools

**Moral defectives**, that is to say persons in whose case there exists mental defectiveness coupled with strongly vicious or criminal propensities and who require care supervision and control for the protection of others

These definitions may be further elucidated by pointing out that —

**Idiots** cannot speak well have a very limited understanding cannot find their way about in the house or in the street are incapable of work of any kind and have to be washed and cared for and even fed, like children

**Imbeciles** can be taught to understand and to do simple tasks though they need to be watched over in washing and dressing They can manage to find their way about the house or school but not usually in the streets

**Feeble minded persons** can be taught to do a varied range of poor grade work, and sometimes can learn to do a single thing in a more expert manner They can be taught a little reading and arithmetic but are incapable of planning things out for themselves or of managing their own earnings

**Incidence.**—It has been estimated<sup>1</sup> that there are 8 per thousand mental defectives in the population In the rural districts this is 10·4 per thousand and in the urban districts 6·7 per thousand In each 100 defectives there are —

5 Idiots,  
20 Imbeciles,  
75 Feeble minded

It is estimated (Lewis 1929) that the incidence of mental defect has doubled since 1907

**Sex incidence**—There are 9 male to 8 female defectives

**Ætiology**—(a) **Primary amentia** (Congenital or inherited)—This group makes up 80 per cent of all cases Among the various factors is *heredity*, Tredgold<sup>2</sup> and others consider this extremely important They argue that if you mate two subnormal individuals you are bound to have subnormal children If one parent be subnormal, 50 per cent of the offspring will be subnormal Tredgold thinks that from 5 to 15 per cent of the parents of mentally defective children are themselves subnormal Other authorities (McNeil 1931)<sup>3</sup> do not agree

(b) **Factors producing secondary amentia** (acquired or environmental)—This group makes up the remaining 20 per cent

(1) *Before birth*—Alcohol, metallic poisons infections, endocrine disturbances deficiency diseases, and anaemia

(2) *During birth*—Birth injury or trauma from head moulding pressure, and asphyxia Prematurity also plays a part

(3) *After birth*—(a) Trauma, as from accidents and falls, (b) infections such as cerebro spinal meningitis and various forms of encephalitis,

<sup>1</sup> L. Findlay "Mental Deficiency" *Lancet* March 5 1935 1 539

<sup>2</sup> A. P. Tredgold "Mental Deficiency" Baillière Tindall & Cox 6th Edn., 1937 Report of the Mental Deficiency Committee 1929 H.M. Stat. Office

<sup>3</sup> C. McNeil "Heredity a Minor Factor in Mental Deficiency" *Br Med Jour* March 31 1931 1, 161

(c) convulsions probably due to epilepsy causing small punctate hæmorrhages into the brain (d) encephalopathies such as those due to lead (e) errors of metabolism as in the lipoidosis (Tay Sachs disease) (f) sensory deprivation as seen in deaf and blind children (g) endocrine deficiencies as seen in the cretin

**Diagnosis.**—This depends on comparison with the milestones of the normal child —

*At birth* — A child shows only reflex actions such as crying sucking and blinking. These are reactions to outside stimuli.

*At 1 month* The infant should be able to follow a bright light with the eyes.

*At 2 months* A definite interest is shown in surrounding objects.

*At 3 months* He shows his emotions by pleasurable sounds and smiles screams or rages. He recognizes his mother and can lift his head from the pillow.

*At 5 months* He can grasp objects with his hands and recognize his parents' voices.

*At 6 months* He can sit upright and change an object from one hand to the other.

*At 9 months* He makes a definite effort to crawl. He is tremendously interested in his environment and continually handles all objects in his vicinity.

*At 1 year* He can stand and take a step or two. He can say single words and obey simple commands. He can drink from a cup and unscrew the top off a bottle or unwrap the paper covering from a sweet. He gives evidence that he experiences the emotions of rage fear joy affliction jealousy disappointment and anxiety (Fredgold).

*By 2 years* He has learnt a great deal as he has been walking for nearly a year. He can now say sentences of several words and has a vocabulary of two to three hundred words. He wishes company and playfellows. He may know some of his colours. During this year he may be autocratic and self-willed and wish to dominate those about him. He screams when thwarted and it is difficult to reason with him. He will pull a stool over to an object which is too high to obtain from the ground. He has now gained control over the bladder during the day, and very often at night. He has complete control over defecation.

*By 3 years* He can say nursery rhymes, knows his colours, and the names of a large number of objects. He can count a little and knows several of his letters. He appreciates signs, and knows which is the longer of two lines. He can make drawings of familiar objects, and plays nicely with toys, showing much make-believe and fantasy in his play (Fredgold). He is now old enough to be reasoned with and is gaining self-control, and is much less difficult to manage because of this. Kindergarten school is of the utmost importance in his life at this age, as it fetches him co-operation.

*By the fifth year* he knows his letters, and can count up to 10 or more.

By the 6th year he can read short words, and from then on his learning is largely determined by the interest his school can arouse in him. By the 7th or 8th year he can reason and form judgments, and can profit by his experience (Tredgold). His play is much less make believe, and he likes school games.

By the 9th or 10th year he develops hobbies and is keen on games, and can reason for himself.

By the 13th year he can adapt himself in an intelligent manner to new situations as they arise in life, and he shows a considerable degree of intelligence, wisdom, and general knowledge.

At puberty there is a tendency to emotional disturbances, but after this the mental powers forge ahead.

At the 16th year the development of the purely intellectual processes of the mind has attained completion (Tredgold).

In diagnosing the mentally defective child, various factors should be noted.

(1) *Delay in acquiring motor control*—The infant may be slow in following a light sitting up, or walking. He may be late in acquiring speech and control of the sphincters. There may be various forms of spasticity and ataxic movements suggesting some cerebral lesion.

(2) There may be *abnormalities of conduct or behaviour*. Undue screaming and purposeless repetition of abnormal movements, should attract attention.

In older children fits of uncontrolled temper, or a disinclination to play properly, or to respond to the environment, are of significance. Undue friendliness is often characteristic of the mentally defective epileptic. Moral obliquity may show itself later in childhood.

(3) *Abnormalities of appearance*. The head may be unduly large or small. The eyes may show the slanting tilt of the mongol, with its exaggerated inner canthus. There may be *stigmata of degeneration*, as seen in the low forehead, badly developed lower jaw, or abnormally shaped skull.

(4) Some of the *special senses*, such as sight or hearing, or power to produce sound may be absent.

Lesser degrees of mental defect may be recognizable only when the child has commenced school, and they then become obvious when he is contrasted with his school fellows.

## SIMPLE PRIMARY AMENTIA

### (CONGENITAL OR INHERITED)

**Incidence.**—This forms the commonest group of all defects, and makes up 73 per cent. of the total (Tredgold).

**Pathology.**—On the whole, the brain is smaller than the normal, although this is not a diagnostic point. There may be agenesis of the cerebral tissues, particularly the cortex. The convolutions are crude and poorly formed. Microscopically, localized areas of hypoplasia of the various layers of the brain tissue and cells are seen. There is a numerical

deficiency, imperfect development, and irregular arrangement of the constituent nerve cells, especially in the pre frontal and parietal areas (Shrubsole)

**Clinical picture.**—Such an individual may appear normal, or almost normal, and it is only from abnormalities of conduct and backwardness in reaching the various milestones that mental defect is suspected. On the other hand, he may have such a gross cerebral lesion that he takes no notice, and is in a world of his own. Such children may fall into the category of "idiots," or the defect may be so slight that it may be termed "feeble mindedness" or "moral defectiveness." The primary ament may be late in acquiring cleanly habits, and show abnormalities of behaviour. He may make unusual sounds, or movements with the body or hands attracting attention. Occasionally he may show stigmata of degeneration, e.g., of the skull and eyes.

#### MONGOLISM

**Incidence.**—This makes up 5 per cent. of the total of mental defectives but 50 per cent. of those mental defectives noted during the first year of life (Tredgold). Very occasionally a second mongol may occur in a family.

**Ætiology.**—This is not known.<sup>1</sup> First there is a high incidence in elderly mothers, at the end of the child bearing period. In 211 cases, the mother's age was thus distributed —

20-24	4 cases	35-39	70 cases
25-29	11 "	40-44	84
30-34	22 "	45-49	20

Secondly, in a high proportion of the parents, the father is younger than the mother.

**Pathology.**—The brain is rudely small, with simple convolutions. Microscopically there is defective neuronic development similar to that in simple primary amentia. The endocrine system is not involved. Congenital heart disease is present in 15 per cent. of all cases.

**Clinical picture.**—The clinical picture is characteristic almost from birth. The eyes are slanting, with an exaggeration of the inner canthus. Blepharitis is extremely common. The child is given to sucking the tongue, which tends to protrude from the mouth, and is inclined to be slim and pointed at its tip, and in time shows furrows on the dorsum.

The head is undersized, all such cases being slightly microcephalic. There is flattening in the occipital region, and often a slight squint, and the whole muscular system tends to poor tone. This hypotonia is best



FIG. 44.—Mongolism showing typical slant eyes.

<sup>1</sup> G. Shrubsole and A. C. Williams, "Mental Deficiency," Centr. of London Press 1932

seen in the arms and legs, especially the hands. A nurse holding the child will remark that it lies in her arms like a bag of jelly, instead of the firm, live feel of the 'normal infant'. There is often a tendency to umbilical hernia. The hands are small, the fingers tapering, and the little finger especially is shortened and inclined to curve in. The skin is smooth but tends to roughness in parts which are exposed to the weather. The colour of the cheeks is high. Very often the child is said to be too good, seldom crying.

An older mongol presents many of the characteristics just described (Fig 44). The mouth turns down at the corners. A considerable proportion of mongols have some other congenital malformation. The child is under sized and the progress of growth is as a rule poor. He is slow in sitting up and walking. The mentality may be very deceptive, as he seems bright, takes a great interest in his surroundings, smiles up into the face of his mother and is most affectionate. It is not until he is expected to talk that gross mental defect is suspected. Mongols are, as a rule, fond of music and affectionate and gentle. Occasionally they are destructive.



Fig 45.—The hands in mongolism. note short incurved little finger

Imitation is highly developed, initiative is lacking. They can be taught to be clean in their habits and moderately obedient, but they cannot be trusted to any great extent and do not prove useful members of society. Institutional treatment has a beneficial effect. Because of the short nasopharynx, the child tends to make a snorting noise as if adenoids were present, almost from birth. Frequently adenoids and enlarged tonsils do appear later and should be dealt with.

**Prognosis.**—Most mongols die before their twentieth year.

**Treatment.**—There is no rational treatment. Thyroid has absolutely no effect on the mentality of these children, but since there is a natural tendency to improve, thyroid has gained an unwarranted reputation. Most help is gained by concentrating on education and keeping up general

\* Dora M. Perry. An Investigation of Fifty Cases of Mongolian Imbecility from the O.P.D., Hospital for Sick Children, Great Ormond Street, Brit Jour Child Dis 1924 xxi, 259





Plate 11 —Brain of a child ten months old, showing tuberculous masses  
at A, B, and C



Plate 12 — Normal brain (on left) and brain in a case of mental defect (on right). The occipital region in the normal brain (on the left) shows well marked development and projects beyond the cerebellum, while in the other brain there is no such development.

health. Careful mothering, the inculcation of cleanliness and checking objectionable habits are most essential. Help can be obtained from an instructive booklet by Dr John Thomson entitled *Opening Doors* (Oliver and Boyd). Since such a child is liable to catarrhal infections, rickets and other diseases, great care of the diet and attention to tonsils and septic foci are required. Solid food is not taken well largely because of the child's mental condition and fluid nourishment should be restricted so as to encourage him to take his solids. All the vitamins are indicated.

Suitable homes can be arranged in the British Isles by the National Provisional Council for Mental Health, 39 Queen Anne Street, London W 1. The local Medical Officer of Health or School Medical Officer has it in his power to certify such a case and recommend the child for institutional treatment.

#### MICROCEPHALY

Most microcephalics are low grade imbeciles, being either idiots or imbeciles. They make up about 1 per cent of all defective children under 10 years of age, and 0.5 per cent of all defectives at all ages.



Fig. 46.—Microcephaly

**Ætiology**—The cause is not known and more than one microcephalic may be born in a single family.

**Prognosis**—Since convulsions are common in this condition and the children are liable to diseases such as tuberculosis and have a poor immunity to infection, they usually die during childhood.

**Clinical picture**—The head has usually a circumference of less than 17 inches, but slighter degrees of microcephaly are common. The forehead recedes and the head slopes to a point from all sides, rising sharply towards the vertex. The mentality varies greatly.

**Pathology.**—Hypoplasia of the cerebral hemisphere and also of the cerebellum is characteristic, and most marked in the prefrontal, parietal and occipital lobes. Ateas of localized cortical agenesis (microgyria) are present. Microscopically, the cortical cells are defective in number.

**Treatment.**—There is no rational treatment. Education, and attention to the general health with institutional life are indicated.

### TUBEROUS SCLEROSIS (FIBLOIA)

This is a rare condition making up only 0.5 per cent of mental defectives. The syndrome consists of tuberous sclerosis of the brain, mental defect, epilepsy, adenoma sebaceum, usually seen on the cheeks, and tumours of various organs. It is possible that the condition is allied to von Pecklinghausen's disease. The adenoma sebaceum of the cheeks is first seen between the fourth and fifth year. It may occur on the forehead and neck. Such children seldom survive to maturity (Tredgold).

### HYPERTELORISM

Described first by D. M. Greig in the *Edinburgh Med. cal. Journal* 1924, the condition is now well known. The child is mentally backward and presents a characteristic appearance. The eyes are widely set and this great breadth is attributed to that portion of the sphenoid bone which arises from cartilage. The skull is high with a flat occiput. The frontal eminences are prominent and there is a median furrow in the frontal bone. The nose is broad and turned up with a flattened bridge, the palate is high and narrow, the mouth is open and the tongue fissured. Some of the features are definitely mongoloid. The outlook for such children is similar to that of the mongol.

### SECONDARY MENTAL DEFECT

#### (ACQUIRED OR ENVIRONMENTAL)

**Mental defect due to trauma.**—The best examples are seen following birth injury (Little's disease). This is described on p. 224. This group make up 5 per cent of the total of mental defectives.

**Mental defect due to infections.**—A full description of mental defect secondary to cerebro-spinal meningitis will be found on p. 230, and to encephalitis lethargica on p. 227.

### HYDROCEPHALUS (WATER ON THE BRAIN)

An enlargement of the head due to an increase of cerebro-spinal fluid either within the brain (internal hydrocephalus) or outside the brain (external hydrocephalus).

**Ætiology and pathology.** *The non-communicating form.*—In this type the fluid is pent up inside the brain and is unable to escape into the subarachnoid space and spinal meninges where it would be absorbed and find its way back in the blood stream. This may be due to developmental errors such as a congenital closure of the foramen of Monro or the aqueduct of Sylvius. Basal inflammations such as occur in posthæmic meningitis due to meningococcal infection or to syphilis close the foramina of Magendie and Lushka and are responsible for most cases of this form of hydrocephalus. Tumours pressing on and closing the various foramina or on the fourth ventricle also cause internal hydrocephalus. Hemorrhage into the ventricle may occur at birth, blocking the foramina or the aqueduct of Sylvius and thus producing hydrocephalus. This is most common in premature infants, and may account for the history of a normal labour with a normal sized head which rapidly increases in size after birth.

*Communicating form.*—Although in this form the fluid may pass freely from the choroid plexus to the subarachnoid space the mechanism for absorbing and carrying away the fluid may be defective. This is sometimes due to a congenital malformation, but much more often the delicate stomata of the lymphatics have been closed or

occluded as a result of acute or chronic infection. Mild forms of meningitis such as those due to the meningococcus or syphilis, are most often responsible.

Again, the absorption mechanism may be perfect but too great a quantity of cerebro spinal fluid may be excreted by the choroid plexus. This may be congenital, or due to some endocrine disturbance or other cause at present unknown.

From long-continued pressure the brain substance is reduced to extreme thinness. At autopsy in some cases it is as thin as a sheet of paper and does not bear handling. The motor area and other portions are thus partially or entirely destroyed so that mental defect and spastic paralysis are to be expected. The pituitary gland may be atrophied from pressure.

**Clinical picture.**—Labour may be difficult owing to the enlarged head of the infant and may often require its destruction. These cases are of course, congenital. On the other hand, the infant may be born apparently normal and in the first few weeks of life the head may increase extremely rapidly, so that by the age of 3 to 6 months it has attained enormous proportions. The forehead is very prominent overhanging the rest of the face, the eyes being everted and the sclerotics showing above. The fontanelle is widely open, sometimes being 3 to 5 inches across. The sutures are also widely open, and the posterior fontanelle fails to close. The bones of the skull are greatly thinned and on pressure may yield slightly. A powerful lamp placed near the head shows it to be partially translucent. When hydrocephalus develops after post-natal meningitis in an older child however, such an extreme picture does not occur. In the infant, who tends to lie where it is placed in addition to protrusion downward of the eyes, constant lateral nystagmus is frequent. A tendency to vomit and thus soil the pillow is a great danger, as the head is then liable to cellulitis which heals badly. Should the hydrocephalus be mild, a balance is struck between the fluid excreted by the choroid plexus and that carried away by the lymphatics. Thus the head ceases to enlarge. The small body in time gradually catches up in growth to the huge head and ultimately very little if any enlargement is to be noted. Because of the thinning of the brain and the constant pressure on the pituitary and fourth ventricle, the clinical picture may vary greatly. Thus, signs of an upper motor neuron lesion may be present, and spasticity, especially of the legs is common. There is usually also a tendency to put on much flesh, probably due to pituitary damage—the fat child.

**Mentality.**—Some cases show very slight mental defect but the majority are grossly affected, and all show emotional instability, tempers, tears or fits of stubbornness being common. Fifty per cent. of all hydrocephalics suffer from convulsions and this serious complication tends to shorten their lives.

**Diagnosis.**—In studying a case of hydrocephalus it is first necessary to ascertain whether it is of the communicating or non-communicating type. A lumbar puncture or cistern puncture is useful as, if the fluid flows freely and a considerable amount may be withdrawn, it is safe to assume that this is the communicating type. Various dyes have been employed to prove this. For example 1 c.c. of methyl violet solution is injected by means of a very fine needle through the open fontanelle into the ventricle. 15 or 20 minutes later a lumbar puncture should be done and the dye should be recovered in this fluid. If the fluid flows badly from the lumbar region or if the dye fails to appear, it may be assumed that it is a non communicating case, that is there is a block somewhere at the foramen of Monro, aqueduct of Sylvius, or foramen of Magendie.

**Intraventricular indicator.**—Neutral phenol sulphone phthalein (Dandy) (prepared by W. Martindale, 12, New Cavendish Street, W. 1) is used. The lateral ventricle is punctured and 1 c.cm. of the solution in the syringe is mixed with the cerebro spinal fluid by the withdrawal of 2 c.cm. into the syringe barrel. This mixture is then injected into the ventricle. Lumbar puncture is performed 5 minutes and 30 minutes after the injection and the cerebro spinal fluid is run into a test tube with a few drops of 25 per cent sodium hydrate. Recovery of the phenol sulphone-phthalein shows that the hydrocephalus is extraventricular or communicating. Non recovery shows that it is intraventricular or non communicating.

**Injection of air (encephalography).<sup>1</sup>**—For some time these cases have been investigated by withdrawing the cerebro spinal fluid by lumbar puncture and injecting air up the spinal canal to replace the fluid both within and without the brain. A ray

<sup>1</sup> Members of the Staff of the Royal Aberdeen Hospital for Sick Children. "Encephalography in the Investigation of certain Cerebral Conditions in Childhood," *Arch. Dis. Child.*, 1936 No. 62, 21, 77; No. 63, 22, 87.

photographs then show the exact state of affairs (Plate 13). Valuable information can be obtained in this way, and with careful manipulation there is little danger. As a rule, within 24 hours the air has been absorbed. In the closed or non communicating form the air must be injected directly into the ventricle, and on this account the method described is less suitable. The needle passing through the brain substance unavoidably damages it, and there is a tendency to bleed. With skilful technique, however, the attempt should meet with success but it is not recommended otherwise.

**Treatment.**—Before any form of treatment is considered, a Wassermann should be done on the cerebro-spinal fluid of all cases of hydrocephalus, to exclude syphilitic meningitis which answers to anti-syphilitic treatment.

It has recently been suggested that wheat germ oil (Vitamin F) is successful in treating communicating forms of hydrocephalus.<sup>1</sup> The dose suggested was a mixture of wheat germ oil (Eli Lilly & Co.) and Vitamin B Complex (Lederle Laboratories) one part of oil to four parts of Vitamin B Complex. The initial dose was 4 c.c.m. of this mixture three times daily for children 2 years and over and 2 to 4 c.c.m. daily for infants, given in the feeds. Scarff<sup>2</sup> reports endoscopic cauterization of the choroid plexuses with several recoveries.

Various operations have been devised for non communicating forms of hydrocephalus (see *Surgery of Childhood* (Arnold) by Sir John Fraser).

Syphilitic meningitis and cerebral syphilis are described on p. 319.

### TOXOPLASMOSIS

This has recently been recognized as a disease of human beings, and it is caused by the organisms of the genus *Toxoplasma* which are protozoan parasites with distinct cytoplasm and nuclear chromatin. This infection produces an intercal hydrocephalus in infancy with choroid retinitis and convulsions. Later X-rays show small areas of calcification throughout the brain. Destruction of the brain may be demonstrated by ventriculography. An acute hydrocephalus appears shortly after birth, lumbar puncture yields a slightly cloudy, xanthochromic fluid which clears promptly, and contains an increase of white cells, mainly lymphocytes with an increase of protein.

The diagnosis is made by demonstrating organisms in the cerebro-spinal fluid and in the brain tissue, or the fluid may be injected into mice, guinea pigs and rabbits, which are susceptible, or serological tests may be done.

**Treatment.**—It is not yet known whether the Sulpha drugs are effective or not.

### AMIAUROTIC FAMILY IDIOCY (TAY SACH'S DISEASE)

**Etiology and pathology.**—This is a familial disease confined for practical purposes to Hebrews and showing the first signs in infancy. The characteristic changes are seen in the ganglion cells of the nervous system—these cells are ballooned up with lipid material and under the microscope have a clear appearance, with the nucleus pushed to the side. The changes are widespread and are found in the cells of the cortex, ganglia and cord. The ganglion cells of the retina are similarly affected, and this accounts for the cherry red spot seen in the macula. The condition is thought to be due to an inborn error of lipid metabolism and perhaps is allied to Niemann-Pick's disease.

**Clinical picture.**—An infant a few months old who may have shown signs of being normal and taking notice begins to fail and ceases to be aware of its surroundings. Soon it is obviously blind and mentally defective. There is, as a rule, some rigidity and spasticity. The picture is like that of a spastic diplegic.

An examination of the fundi shows a pale area in the macula and in the centre of this a cherry red spot which is pathognomonic of the disease.

**Prognosis and treatment.**—The child dies at about one to two years. There is no treatment.

### CRETINISM

For an account of this condition, see Chapter XX, p. 367.

<sup>1</sup> S. Stone: Wheat Germ Oil (Vitamin F) in the Treatment of Congenital Non-Obstructive Hydrocephalus, *Jour. Ped.* 1943 12:11, 191.

<sup>2</sup> J. E. Scarff: "Non-Obstructive Hydrocephalus—Treatment by Endoscopic Cauterization of the Choroid Plexuses," *Amer. Jour. Dis. Child.*, 1943 12:11, 297.

<sup>3</sup> A. B. Cabot: "Toxoplasmosis, Advances in Pediatrics, Vol. 1 Hanesman 1941"



Fig 1 —Lateral ventricle



Fig 2.—Front view showing both lateral ventricles.  
Plate 13 —Pneumoradiograms showing air in  
the ventricles of the brain

## EPILEPSY

This group makes up 1 per cent of all mental defectives (Tredgold)

**Ætiology.**—Epilepsy may begin at any age. The diagnosis under the age of two years is difficult because symptomatic infantile convulsions such as those secondary to rickets, breath holding and infections, closely resemble those of epilepsy. At least one half of all epileptics have convulsions before the age of ten years. The commonest ages at which convulsions appear are with the second dentition namely six years, and at puberty.

**Pathology.**—Epilepsy as a disease probably does not exist. It must rather be looked upon as a symptom. It is convenient however clinically, to group certain syndromes under the name of epilepsy.

Probably in the vast majority of children who have convulsions certainly after the first dentition is complete there is some organic cerebral lesion. The introduction of the electro-encephalogram has demonstrated cerebral scars and areas of degeneration which have been confirmed at operation. Birth injury and mental defect with malformations must account for the majority of epileptics.

In children who have had a large number of fits, over some years much sclerosis, mingled with areas of cerebral softening is found *post mortem*. It is probable that, with each convulsion small punctate hemorrhages occur in the cerebral tissue, which later give rise to these changes.

**Symptoms.**—As a rule the first attack may occur in what has been considered a perfectly normal child. The convulsions may be either grand mal or petit mal. In grand mal the stages of the fit are—

(a) *Aura*—The child may complain of an unpleasant or unusual taste, smell, visual disturbance or sensation. The same aura precedes each convulsion. The aura may last for a few seconds or longer, and is followed by (b) *loss of consciousness*. Usually the child falls down, or may have had the presence of mind to sit or lie down during the aura. Almost immediately after this (c) the convulsion occurs. This has a tonic or stiff stage, in which all the muscles are rigid and the eyes are open and staring, the breath being held and the face becoming cyanosed. After a varying period the clonic or shaking stage sets in and this may last for a few seconds up to several minutes. During this phase there is frothing at the mouth, and the froth may be blood stained if the tongue has been bitten. The sphincters frequently relax, so that the child passes its urine and occasionally feces. Usually (d) *relaxation and sleep* follow, but in a few minutes or longer the child rouses and may be quite normal, or there may be confusion for some hours afterwards.

*Petit mal*—These are often called "little turns" or "absent moments" and many dozens may occur in the day. Such turns are often missed or put down to "nerves". They may last for a second or two or at most a few seconds. The child seldom falls. Often, a pause in the conversation with a fleeting appearance of bewilderment over the face and eyes, is all that takes place. Occasionally, there is a sudden twitching of some part, such as the shoulders or face, or there may be a jerking forward of the head and body in a sudden spasm. This is characteristic of the salaam type of epilepsy in infants.



were electrical potentials in the living brain cortex. Berger demonstrated in 1934 that electrical potentials arose from the nerve cells of the cortex, and he called the potentials which varied in frequency from 1 to 60 per second with a 10 per second rhythm 'alpha waves' where as those with a frequency of 15 to 60 per second were 'beta waves'. Alterations in activity (dysrhythmias) were discovered in persons suffering from epilepsy.

The normal electroencephalogram of children is slower than that of adults and it increases in frequency with age. At four months it may be only 4 waves per second, whereas at three years it is generally more than 7 per second, reaching 10 per second at the age of 10 to 15 years. Once the adult pattern of activity is reached waves slower than 8 per second do not normally occur (the 'alpha' activity may vary from 8 to 12 per second). With any disturbance in brain function, such as that caused by an expanding lesion, brain degeneration or epilepsy, slow waves make their appearance. Slow waves may be produced by hyperventilation when waves as slow as 3 per second could be elicited in epileptics. A high proportion of children suffering from behaviour problems show abnormal electroencephalograms, particularly bilateral asymmetry, i.e. different frequency on the two sides of the brain. Definite correlation between the type of behaviour disorder and the electroencephalographic abnormalities is not yet possible.

Slow frequency waves and bilateral asymmetry found in those above infancy suggest functional abnormality of the brain. Nevertheless it is not possible to draw sweeping conclusions from this form of examination. The electroencephalogram is a help to the clinician only in demonstrating dysrhythmia of brain function.

**Treatment. Drugs**—Bromides, particularly in the form of syrup of bromocarpine, are useful, and the dose should be from 60 to 180 grains twice daily, dependent on age.

The barbiturates, such as luminal or prominal, are extremely popular. The usual dose for a child of from 4 to 12 years would commence with half a grain night and morning.

Recently Putnam and Merritt<sup>1</sup> have found that a drug *Epanutin* (sodium 5:5' diphenylhydantoinate) (Parke, Davis & Co) is a strong anti convulsant. This has been given to children with varying success. The object of this rather toxic preparation is to block, or cut off the convulsions. It is not, therefore, a sedative, and the child is not made dull or stupid while taking it. In about 40 per cent of epileptics<sup>2</sup> the results are excellent. There is diminution or cessation of the attacks, but after a time a proportion relapse. It is, however, the most satisfactory drug available at the moment. About 15 per cent of the children develop toxic symptoms, and have to discontinue it. These are shown by a morbilliform rash, tremors of the hands and legs and giddiness. The usual dose is from one to three grains in the twenty four hours but, as a rule, three are required.

Often it may be found best to give *Epanutin* along with barbiturates, and a very satisfactory combination for infants is—

R. *Epanutin* ½ grain  
I luminal, ¼ grain

<sup>1</sup> Putnam & Merritt, "Arch. Neurol. and Psych." Chicago 1935 xxix 1003

<sup>2</sup> Halse Bailey & McGregor "Treatment of Epilepsy with Epanutin." *Lancet* Aug 1935 ii 262

organic disease as the probable cause of many behaviour problems. At least, he must keep a very open mind in this respect. For example, when a child has been particularly naughty, and before any punishment is meted out, it is often wise to take the child's temperature. It may be found that the loss of control over conduct is produced by the onset of an acute infection.

### NERVOUS EXHAUSTION

(NERVOUS FATIGUE)

The appearance of the so-called nervous exhaustion child has been well described by Hector Cameron.<sup>1</sup> This child is pale, thin, full of energy at one time, and utterly exhausted at other times. The stance is characteristic of fatigue: the shoulders droop, the pelvis is pushed forward, and there is a tendency to be round-shouldered, with the shoulder blades sticking out. All the joints may be hyperextended and the muscles are hypotonic. The face muscles seem to be ironed out and expressionless. When exerting himself he has a good colour, but in repose his colour may leave him almost entirely, giving rise to what are called "pale turns." Children to whom this description applies are subject in greater degree than the average child to nervous and metabolic disturbances and are unduly upset by passing infections.

Cameron has stressed the need in such children for an increase of sugar in the diet, particularly in the form of glucose. Their activity is so great that they tend to burn up their carbohydrate and in turn call on fats with the production of ketosis.

More often than not, it is a passing infection, such as tonsillitis, which initiates the ketonæmia. A description of such attacks is given on page 123, under the heading of "cyclical vomiting" or "bilious attacks." A liberal allowance of sugar is undoubtedly of benefit in such cases, both as prophylaxis and treatment. Fat should not be unduly restricted, but attention should rather be directed to removing the source of infection, if possible.

**Treatment and management.** *Rest*—Usually these are only children, or the youngest by several years, and are too much thrown into the company of adults, thus being constantly on "mental tiptoes." Much benefit is derived from early kindergarten school, even at the age of three or four years. Here a child is taught to sit still, relax, and play along organized lines; mental and physical rest is obtained. If this is not possible, the child should spend the morning, as far as can be, off his legs, in a pram or elsewhere, and a rest at midday is essential.

**Understanding supervision**—Those in charge of the child should be aware of his tendency to become easily exhausted, and should always be on the look out for signs. As a rule, it will be shown by deterioration of conduct.

<sup>1</sup> H. C. Cameron. "The Nervous Child." Oxford Medical Publications.



Fig. 47—Nervous-exhaustion child showing position of general hypotonia. (*British Medical Journal*)

may prove to be considerably over weight for the age and height. The refusal is simply a defence mechanism against further overweight. If this be pointed out to the parents, insistence on finishing each meal will cease. Cutting down starches, such as cereals, milk puddings, potatoes and bread, also benefits such children.

To summarize — In attempting to set right a case of anorexia, organic disease must first be excluded. This may be done by a careful examination of the throat, mouth and other organs always including the urine. Next, the diet must be considered to see that it is well balanced and that, while it furnishes the necessary vitamins, it is not too exclusively made up of cream and fatty foods.

A change of air and surroundings may be necessary, possibly a change of nurse, or a visit to a relative or friends or from day school to boarding school or convalescent home.

**Tonics** — Iron containing tonics such as the following are indicated —

As much sugar of iron as will be on a sixpence, three times a day after meals.

Or

Iron and ammonium citrate, 5 grains

Syrup 30 minims

Water to 60 minims

One teaspoonful three times a day after meals

Liver should be introduced into the diet once or twice weekly. Cod- or halibut-liver oil should be given in suitable forms such as with malt, and vitamin B, given as brewer's yeast, promotes appetite.

**Anorexia nervosa in older children.**—One of the serious complications of puberty, particularly in girls, is that of severe loss of appetite. Such children show no organic disease. Their mental outlook towards their surroundings including parents, relations, food and life in general, is abnormal and psychopathic, with marked depression. They cannot be roused to take an interest in anything and, if left alone, may die.

**General management and treatment.**—It is best to remove the patient from home surroundings. Often the child has developed a mother complex, and this situation can best be dealt with in a hospital or nursing home where cheerful and optimistic persons surround her. Parenteral crude liver extracts should be given, such as Proethiron forte (Armour) 2 cc on alternate days alternating with Vitamin B Complex, 3 cc (Lederle) also parenterally. A diet high in carbohydrate and containing malted milk is particularly suitable. Once the child has commenced eating all the vitamins should be added and a general diet instituted. Massage, artificial sunlight and iron tonics should be given. Ross's case<sup>1</sup> aged 12 years originally weighed 39 lbs only, but gained 27 lbs in 58 days.

Fat children in an effort to slim may relapse into this condition, and it should be recognized that slimming carries this danger.

## DISTURBED SLEEP AND NIGHT TERRORS

**Ætiology.** In the infant — Nasal obstruction from a cold or catarrh is probably the commonest cause of broken sleep. A drop of liquid

<sup>1</sup>G. W. Ross, "Anorexia Nervosa with special reference to the carbohydrate metabolism," *Lancet*, May 7 1935 1, 1017.

paraffin down each nostril, or a drop of glucose solution (Parke, Davis & Co.), will help to clear up the nasal passages. They may be cleared mechanically with pledgets of cotton wool, or the infant may be made to sneeze.

*Over feeding*, especially when the child is not being held up afterwards to break or bring up the wind, tends to cause colic and indigestion and with each bout of colic during the night the child tends to cry out.

*Under feeding*, with consequent hunger pangs, will likewise disturb the infant. An adequate feed will cure this.

*Neuralgia during teething*—Although there is no scientific evidence to support this supposition, it is a remarkable fact that a great many children who have never had a complete night's sleep during primary dentition sleep soundly after cutting the last tooth.

*Over or under clothing*—Each child is a law unto itself. Hot children require very little bed clothing, and "cold" children may require bed socks and hot water bottles if they are to sleep the night through.

*Older children—Insufficient fresh air and exercise*—Children living in indoor life may have had insufficient oxygen to metabolize their food during the day. Such children sleep more soundly after a full day in the fresh air, with outdoor exercise. Late sleeping in the morning should be discouraged.

*Bed wetting may wake a child*, as he is cold and uncomfortable.

*Mental ulcer*—This condition often develops as a result of bed wetting. Each time the urine is passed the child wakes because of painful micturition. Treatment is outlined on p. 206.

*Fear*—Some children are fanciful, and see in the shadows frightening objects. Such children should be encouraged to speak out, to reassure themselves, and should be given a night light temporarily. The door of the bedroom should be left open, so that they can hear others passing by.

*Attracting attention (exhibitionism)*—Some children call out in the early part of the evening, and will not settle off without several visits from the parents or nurse. Very often this follows the arrival of the new baby. If this be the case, the new infant should be placed asleep in the room with this child and left in his charge. He must be told to be quiet or he will wake the baby. He should be allowed to have his particular toy or plaything. Firmness and understanding will get round this short phase.

*Constitutionally sleepless children*—Some children go to sleep promptly, but wake either very early or during the night, quite refreshed and playful and do not appear to require further sleep. Their day should be gone into to see whether they are having too much rest, and insufficient exercise and fresh air. If necessary, they should be put to bed a little later. If awake in the night, they should be attended to, but if dry and warm they must be very firmly put down, and it must be made plain to them that "this is not time for play". Such children are sometimes better in a room by themselves, where slight noises will not wake them if they be light sleepers, and where, once awake, they can croon themselves to sleep again without disturbing others.

*Over stimulation of the brain before going to bed*—Some children are read

to and played with during the last hour before bedtime. This may so 'work them up' and stimulate them that it takes some time for them to become placid and quiescent enough to go to sleep. Such stimulation should be stopped.

**Drugs** — *Chloral hydrate* is the safest drug during infancy and childhood and it may be given in doses of half a grain from birth onward, three quarters of a grain at about 5 months, one grain at 9 months, and  $1\frac{1}{2}$  grains at one year. Add one grain for each year after this up to the age of 5 years, when it is better to repeat the single dose rather than give a bigger one.

*Amytal* (Lilly) — One quarter of a grain will be found useful in children aged one year and gr  $\frac{1}{2}$  for those of five years and upwards.

**Night terrors** — The following are some of the commoner causes —

*Nasal pharyngeal obstruction* with enlarged tonsils and adenoids. This is a common cause and removal of the obstructing lymphoid tissue will cure the attacks.

*Digestive disturbance* due to too large or indigestible a supper is a common cause. An earlier tea supper should be instituted and cheese or other indigestible food omitted from the meal.

*Hypoglycæmia* — Cameron quotes cases which are cured by the administration of glucose before going to bed.

*Epilepsy* — It should be borne in mind that attacks resembling night terrors may actually be convulsive seizures.

### NERVOUS VOMITING

Before a diagnosis of functional or nervous vomiting is made, all organic causes must be excluded.

**Organic causes of vomiting** — *Cerebral lesions* such as birth injuries, cerebral tumour and meningitis.

*Alimentary tract lesions* such as obstruction by volvulus, appendicitis, pyloric or duodenal stenosis, peritoneal bands, cardiospasm, oesophageal stenosis, and enlarged tonsils and adenoids.

*Toxic causes* including certain drugs such as ipecacuanha and the effect of acetonaemia on the vomiting centre.

*Digestive causes*, such as gastritis or indigestion from unsuitable food.

*Mechanical causes* including vomiting induced by finger sucking or the coughing of whooping cough, where retching is induced by the mechanical force of the cough, or mucus in the pharynx. Infected adenoids and the post nasal drip of an antrum will cause early morning vomiting also due to the mechanical collection of post nasal mucus.

Vomiting may follow the first introduction of solid food, the larger particles impinge on the tonsils and uvula and thus induce retching. It may be necessary in such cases to sieve all food very finely for a longer period until this extreme sensitiveness has passed off.

### FUNCTIONAL VOMITING

**In infants** — The best example of this is seen in rumination (p. 117).

**In older children** — Some children appear to be able to vomit at will.

and it is characteristic of them that they seldom vomit over themselves, or unless there is an adult present. In such cases there has originally been some organic cause, such as tonsillitis or gastritis, and too much notice has been taken of this episode, and the child has quickly learned that he can attract attention by vomiting.

**Treatment.**—The mother and nurse must be taken fully into the confidence of the physician, and it may be necessary to give a bismuth meal, and have the patient screened in front of the mother so that she can see the bismuth passing rapidly through the stomach and thus be fully convinced. Reassured, she then manages the problem of vomiting with a firm hand, and studiously ignores it.

## MASTURBATION

**Age incidence.**—In females, from the sixth to eighth month onwards. In males, uncommon before the third year.

**Clinical picture.**—Masturbation is extremely common and has been indulged in by almost every individual at some period of life. Female infants and children rock back and forth rubbing the thighs together or crossing the legs. Pressure may be exerted against some object such as the corner of a table or chair. An orgasm is obtained in quite young infants they become red in the face, breathing rapidly and afterwards perspire and become pale and exhausted.

Males may handle the parts at any age but orgasm is rare before the age of five or six years.

**Ætiology.**—This should be looked upon as a reflex act of a pleasurable nature, on a par with thumb-sucking. The child has discovered it by chance, and persists in doing it. Sex does not enter into the question at all until the child is much older. Irritation of the vulva or penis by adhesions or inflammation from want of cleanliness, may attract a child's attention to the genital organs. In males a pinpoint meatus may be a contributing cause.

**Treatment.** *Local and general management.* A careful examination should be made to exclude local irritation. In the female the vulva may be inflamed from want of cleanliness, ammonia dermatitis or adhesions of the labia. Occasionally, thread worms pass forward and cause irritation. In the male, an overtight prepuce or slight inflammation may be present. Sitz baths for the female child, with a little Condy's fluid (sufficient to turn the water pink), and circumcision in the male child will often set the matter right.

Where no local trouble is found, the child should be carefully watched at his resting period, at bedtime, or on waking, as these are the times when the practice is most common. The child's mind should be kept diverted from the generative parts at such times by some artifice, such as giving him a toy to hold. The author has known cases cured by giving the child a spool and rubber band so that he employs his mind by winding the band round the spool, and unwinding it again, until he falls asleep naturally.

Mechanical appliances have not been a success on the whole, although in

a few cases a rolled up towel fastened between the knees of a female infant will prevent thigh rubbing

*Drugs*—Amytal, one quarter of a grain, or luminal, one quarter to one half a grain, given one half hour before bedtime, may induce rapid sleep for a few nights, and cause the child to forget this habit

*What to tell the mother*—The fear in the mind of the mother or nurse is that the act shows a tendency to excessive sexual precocity or some vicious proclivity. This fear is quite unfounded and there is no question of sin entering into the act at all at this age and no permanent harm will result

There is a widespread belief among the public that masturbation leads to mental defect but this is of course quite unfounded. Masturbation is extremely prevalent, and it occupies a short or a long phase in the life of a child depending on his mental make up and on the fuss made about it by the parents. Where those in charge show great anxiety and remonstrate with the child the habit will probably persist for a considerable period. On the other hand where very little notice is taken and the child is kept healthily employed he will pass quickly through this phase. It must be made clear to the child that this is a dirty habit equivalent to picking the nose and that it is *not* approved of and left at that.

*Older children*—Before the onset of puberty a suggestion to the child that he will not be able or fit to do well at his sports together with allowing a minimum of opportunity for masturbation is usually successful. In schools where one individual delights in teaching others the practice that individual should be isolated rather than expelled, and if the situation is cleverly handled he may be made into an ally of the head master. Too serious a view should not be taken before puberty.

### THUMB SUCKING

The evils of thumb sucking have been much over rated and at worst it can only be regarded as a rather dirty habit. In the first few weeks of life the child's thumb or fingers may have been placed in its mouth to ensure that it remains quiet especially during the night and the habit is quickly established. In little children thumb sucking may be practised throughout the day and night but in older children it is done as a rule only when they are tired or going to sleep. Results such as protruding teeth adenoids, nasal obstruction and a high arched palate are all attributed to thumb-sucking but there seems very little evidence for them. In fact many thumb suckers appear normal in every way and many children with adenoids have never sucked either their thumbs or a dummy. The chief objection is the possibility that the child may infect himself with thrush or some pathogenic organism. The best treatment is to substitute some other habit or to distract the child's attention from the one under discussion. In infants it is sufficient to put cotton gloves on the hands or to bandage them, as the child quickly forgets. For older children a variety of apparatus has been devised. The child can often be 'shamed' out of the habit, or the thumb can be painted with some disagreeable substance, such as arnica,

each night. The author's feeling is that this habit is of little practical consequence and that it is wrong to take too much notice of it.

### STAMMERING<sup>1</sup>

In young children at the age of three or four years stuttering or stammering may be looked upon as almost physiological as it is so common. Nearly all children go through a phase in which the desire to speak is so acute that the words seem to rush out and almost choke the child so that articulation ceases. No notice should be taken and in a few weeks or months this phase will pass.

In older children, say at five or six or seven years true stammering, however, is of great importance and requires to be handled with skill if it is not to remain a permanent and a great disability.

**Ætiology.**—Stammering should be looked upon in childhood as a symptom of nervous exhaustion. The child has lost confidence in his power to speak properly. There is frequently an hereditary tendency to stammer or to other signs of nervous exhaustion.

**Clinical picture.**—Such a child seldom stutters at the beginning of the day, but as the day progresses it becomes worse and worse. When playing with children or singing in class he is all right but when singled out to speak individually all his confidence leaves him. It is when he is self-conscious that his stammering is worst.

**Treatment.**—In the early phases the disability should not be pointed out to him. The question of rest should be gone into and if necessary a complete change of surroundings arranged. Tonics should be administered. He should be asked by some individual he knows well to read aloud from a book so that his self confidence will return.

After the age of seven or eight years it is necessary to attempt the voluntary method of treatment. The services of an elocutionist and gymnast are required to teach the child the correct way consciously to phonate and breathe in order to overcome this habit.

It is essential throughout to surround the child by optimistic people, and never to place much stress on his disability. Brothers and sisters should be asked to encourage him and not to aggravate his tendency.

<sup>1</sup> H. St. J. Ramsey. "The Stammering Habit: Corrected through Speech Re-education." *Practitioner* Dec. 1937 xxxix 407. Editorial "Stammering," *Lancet* Jan. 5 1938 i 41.



## CHAPTER XIII

### CONVULSIONS IN INFANCY AND CHILDHOOD

#### CONVULSIONS IN INFANCY

THE diagnosis of convulsions if they are seen is comparatively simple. Unfortunately by the time the practitioner is called, the convulsion or fit is usually over and he must make his diagnosis from the description. Screaming attacks from wind may be mistaken for a convulsion and so may masturbation. On the other hand, *petit mal* may be mistaken for a peculiar habit and the *salam* type of epilepsy in an infant for windy "pneums".

Whenever a practitioner is called to a child with a convulsion, he should make a rule of always *taking the temperature*. This will repay him by helping in the differential diagnosis between epilepsy (which is afebrile) and some of the causes of convulsions mentioned below, which are febrile, and have a better prognosis.

**Description of a convulsion**—This is usually ushered in by a general stiffness of the whole body, the eyes are fixed and are usually widely open and the arms are stretched out with the fingers clenched. The stiffness may last for a few minutes or even for several hours. The child often foams at the mouth. During the stiff period it looks a bad colour, becoming either blue or extremely pale. After this stage there are movements of the limbs and the whole body—the so called *clonic stage*. The fit then passes off and the child relaxes and may pass into a deep sleep. He may or may not have passed urine or stool.

If the fit is a slight one (*petit mal*) the whole thing may be over in a few moments with only dropping of the head and a dazed look in the eyes to mark it.

**Ætiology.** (a) **Congenital malformations of the brain**—In this group are found infants with microcephaly, hydrocephalus and other gross cerebral defects (cerebral agenesis). When the malformation is slight, no suspicion of the defect may be present until the convulsions. In a small infant these may take the form of a sudden bending forward of the body and dropping of the head with the arms stretched out and hands clenched. This is often termed the *salam* type of epilepsy.

Minor infections may precipitate a status epilepticus in a child with gross cerebral malformations. In infants under one year of age, chloral hydrate in appropriate doses (see p. 397) will be found a sheet anchor. Bromide and luminal should be used for slightly older children.

(b) **Cerebral hæmorrhage** (Littles disease with cerebral oedema, birth trauma)—An infant who has had a difficult birth, usually a breech presentation or a premature infant may have convulsions shortly after birth. Usually such an infant fails to suck, cry or breathe well. The

fontanelle may be bulging. A lumbar puncture will show blood stained fluid. Chloral, lumbar puncture and careful feeding, together with hypertonic rectal saline are indicated. For details see p 64

(c) Cerebral infection (encephalitis, meningitis) —A perfectly normal infant or young child develops sudden convulsions with fever, which may last hours, or several days and, when these pass off the child is left mentally backward to a varying degree often showing some spasticity of the limbs. Such a clinical picture may follow an attack of polio-encephalo-myelitis, or encephalitis lethargica. Meningococcal or septic meningitis may be ushered in with a convulsion. The convulsions of tuberculous meningitis are usually late in the disease. The diagnosis is made by examination of the cerebro spinal fluid. The fever differentiates these cases from epilepsy.

(d) Infection outside the nervous system (Symptomatic convulsions) —Some infants and young children usually below the age of three years, tend to have convulsions at the onset of acute febrile disturbances. Tonsillitis, pneumonia, pyelitis and acute infectious fevers such as measles, scarlet fever and influenza, may all be ushered in by a fit. Such children seem liable to a convulsion rather than a rigor when the temperature rises. It is suggested that the convulsions are due to the sudden mobilizing of the chlorides of the blood thus producing profound bio-chemical changes and altered intracranial pressure. Such a group makes up a large proportion of the convulsions seen between one and four years of age. It is possible to predict further convulsions, should this child develop another acute infection. In a series of such cases the average number of convulsions was five, at three or four monthly intervals.

It is seldom that the infection is suspected and the first evidence of ill health is often the convulsion. The originating cause may easily be overlooked in the anxiety of the moment, and, unless the temperature is taken and a careful examination made of the whole child, the disease may go undetected for some time. Usually, however it declares itself in a few hours. No mental defect results from such fits and they cease about the age of four or five years, with no recurrence.

(e) Asphyxia (Breath holding convulsions) —Some children from the age of one to three years, develop a habit of breath holding. They have no enlarged thymus, nor have they spasmodic rickets. When crossed or upset they fly into a rage and hold their breath. They become cyanosed, unconscious, stiff, clonus develops, and they then relax. In a further three or four minutes they are running about as before. This may occur several times a day. These are very alarming turns. It is characteristic that chloral, bromide and other sedatives have no effect.

Such children are usually 'only' or adopted children, or the youngest children of elderly parents. They are strong willed, and lack discipline. The treatment appears to be to educate them to have self control. No bad effects come from these attacks, and they pass off spontaneously about the age of 2½ or 3 years. Sudden application of a cold sponge, at the very onset, will cause the child to inspire and abort an attack. A change of nurse or surroundings, however, is often necessary to bring about cure.

\* Donald Watson \* Insects as a Source of Recurrent Epileptiform Seizures in Children, Arch. Dis Child, No. 41, June 1956, p 135.

In *whooping cough* in infants and children under two years of age convulsions may occur, due to the asphyxia produced by a fit of coughing. The prognosis in such cases is not good and a proportion show mental changes if they survive. The majority, however, recover completely.

(f) *Spasmophilia* or tetany—Since florid rickets is not so widespread as formerly spasmophilic convulsions have become much less common. In spasmophilia the blood calcium is reduced to 5 or 6 mgm. per cent. instead of the normal 10 or 11 mgm. per cent. This reduction of the blood calcium causes an increased excitability and conductivity of the peripheral nerves (for details see p. 78). In such children laryngismus stridulus and convulsions are found. The exciting cause may be an infection, shock, digestive upset, worms or plumbosis.

Chloral should be administered frequently and for the spasmophilia calcium chloride in sufficient doses is specific. Cod liver oil, and a well balanced diet together with real or artificial sunlight are indicated as general treatment.

(g) *Rarer causes*—*Uremia* should be thought of in any case in which the convulsions continue for some time and there is no tendency to recover. An examination of the urine and of the eyes and a blood urea estimation will establish the diagnosis. *Cerebral tumour* and *cerebral abscess* may both be complicated by convulsions. (For diagnosis see pp. 221-222.)

*Tetanus* in infants and children may show itself in the first instance in a series of generalized convulsions. These occur on the slightest stimulation or handling.

*Mumps meningitis* is a rare cause of convulsions.

*Tay Sachs disease* occasionally shows convulsions. (See p. 260.)

*Hypoglycæmic convulsions*—These are said to occur in normal individuals and to be the cause of epileptic seizures particularly during the night. They are said to be held in check by giving glucose at bed time. Hypoglycæmic attacks in diabetics after an overdose of insulin are usually ushered in by faintness, sweating, tremors and loss of consciousness, and may last several hours unless sugar or adrenaline are administered promptly.

*Dehydration* is a not uncommon cause of convulsions and may be seen at the end of an acute gastro-enteritis or dysentery when the infant or child has become excessively dehydrated. Whether this is due to a concentration of the blood with consequent high blood urea, is not clear. In any case making good the dehydration by subcutaneous or intravenous saline and glucose stops the convulsions.

*Poisons* such as strychnine may also cause convulsions.

**Treatment**—Chloral hydrate should be given in doses of half a gram each hour until the convulsions have ceased or the infant is too drowsy to suck properly, when the intervals should be lengthened. At four or five months of age three quarters of a grain of chloral may be given, and at nine months one gram, at 18 months two grains and thereafter one gram added for each additional year of the child's life, up to five years. Small frequent doses are better than large infrequent ones.

**Lumbar puncture**—Whenever there is any suspicion a lumbar puncture should be performed. It will do no harm, and may establish an

early diagnosis from which treatment can commence intelligently. Very often it has a considerable therapeutic value, and the convulsions cease at once.

**Convulsions in older children.**—After the age of five years a convulsion suggests epilepsy, or organic brain disease (*see* p 261).

Hydrocephalics, microcephalics, and spastic diplegics are all liable to convulsions in their older years. Cerebral infections may be excluded by lumbar puncture.

## CHAPTER XIV

### DISEASES OF THE BONES AND JOINTS

#### (A) CONGENITAL DISORDERS

**Hypertelorism.**—This is a congenital and often familial craniofacial deformity giving rise to an excessive width between the orbital fossae. There is usually but not invariably an associated mental defect.

The abnormality is due according to Craig<sup>1</sup> to an overgrowth of the lesser wing and undergrowth of the greater wing of the sphenoid. Unilateral cases have been described (See p. 58).

**Oxycephaly (acrocephaly tower head, steepleskull).**—The head is short from the front backward but is both tall and broad. There is some exophthalmos, and the outer canthus of the eye is lower than the inner. The apex of the skull is in the region of the anterior fontanelle. The skull is thinned and radiographically shows digital markings (Fairbank). The orbits are shallow causing the exophthalmos. Webbed fingers and toes often accompany this condition (acrocephalosyndactyly). Headache is common.

**Hereditary cleido-cranial dysostosis.**—In this rare condition there is a congenital absence of the whole or part of the clavicles. The skull may also be imperfectly ossified in the region of the fontanelle. The teeth and the centres of ossification in the hands, feet and elsewhere, are delayed. Such children can approximate the shoulders in front of the chest.

**Arachnodactyly (Spider fingers).**—In this rare condition the fingers and toes are unduly long and thin. X-rays show that all the bones are likewise elongated and thin. Abnormalities of the iris frequently accompany this condition.

**Congenital elevation of the scapula (Sprengel's deformity).**—The condition is rare. It is commoner in girls than boys. The scapula on one side is small and fixed high up with the inferior angle nearer to the spine (Fairbank). Movement of the arm is restricted.

**Klippel-Feil syndrome (Webbed neck).**—Such children have congenital abnormalities or absence of some of the cervical vertebrae causing the neck to be extremely short. The head is therefore set between the shoulders and there is a fold of skin running from below the ear almost to the tip of the shoulder.

**Marble bones (Albers-Schönberg's disease).**—The bones are unduly dense due to the deposit of much lime. X-rays confirm this. The skull is involved and optic atrophy results from encroachment on the optic nerves. Anemia is present because of the involvement of the bone-marrow. Death takes place from intercurrent disease before puberty as a rule. This condition is rare.

<sup>1</sup> *Graig, Edin Med Jour., 1908 xxxviii 152 and 357*



Fig. 48.—Case of achondroplasia aged 16 height 37 inches

**Achondroplasia** (*Chondrodystrophia foetalis et micromelia*)—This is recognizable at birth. The long bones are shortened, but the trunk is normal. The head is large. The hands are characteristic, as the fingers are approximated in pairs, giving rise to the trident appearance.

The condition may be hereditary, and is due to an interference with endochondral ossification, especially at the extremities of the long bones (Fairbank). Mentally these children are normal. They seldom exceed 4½ feet in height.

**Treatment**—Nothing has been found to influence the condition.

**Congenital dislocation of the hip**—Seven females are affected for each male, and the condition is bilateral in 40 per cent of cases (Griffiths and Mitchell).

The condition is first noticed when the child begins to walk at 12 to 14 months of age with a definite limp, and one hip is seen to be higher than the other.

**Diagnosis**—If the child is placed on the back, abduction and external rotation of the hip are limited on the affected side, and if the knees are flexed leaving the feet on the ground, the knee on the affected side is lower than on the unaffected side. X rays confirm the diagnosis. In bilateral cases the child has a waddling gait and exhibits marked lordosis (Plate 15 facing p. 281).

**Treatment**—This is purely surgical and in most cases proves satisfactory. Seventy-five per cent of cures in unilateral, and 50 per cent in bilateral cases is claimed by Frie Lloyd.

**Talipes equino-varus** (Club feet)—The cause of this is unknown but some

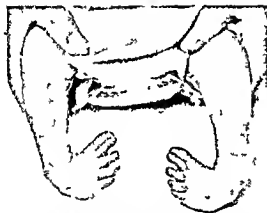


FIG. 49.—Talipes equino-varus.

authorities (Denis Brown) consider it due to pressure on the fetus in utero. The treatment should be commenced as soon after birth as possible, with manipulations and splinting (Fig. 49).

**Fragilitas ossium** (Brittle bones and Blue sclerotics)—This is a familial and hereditary condition (John Thomson). It is rarely diagnosed before the third or fourth year, when fractures begin. At this time the sclerotics are noticed to be intensely blue, and the skull tends to bulge in the temporal region above the ears.

In some cases fractures are infrequent, and the children live a normal life, but others may lead a life of invalidism. Liability to fracture grows less with the years. In adult life osteosclerosis is a common complication. The aetiology is unknown, but there is deficiency of the fibrous trabecula binding the osseous tissues together.

**Blood**—The calcium and phosphorus in the blood are normal.

**Treatment**—No treatment seems of use beyond general care to prevent fractures.

**Osteogenesis imperfecta** (foetal rickets, congenital rickets)—Some authorities (Fairbank) consider this group with fragilitas ossium. The feature is the presence of fractures at birth. (Plate 14.)

**Clinical picture**—The head is often described as feeling "like a bag of jelly" and X rays of the bones show marked osteoporosis. A dozen or more fractures may be present from birth, and fractures may occur in the arms and legs, even when the greatest care is taken.



Plate 14 —Rad ogram in a case of osteogenesis imperfecta  
(same case as Fig 50)



Fig 1 —Congenital dislocation of the hip (left)



Fig 2 —Perthes disease (pseudo-coxalgia)  
(By courtesy of Dr. Heiman S. Reis)



of the joints come together, causing pain. Muscular wasting always occurs in the vicinity of the affected joint.

*X rays* show a fluffy appearance of the articular surfaces of the bones.

**Prognosis.**—On the whole this is good, but occasionally the disease becomes disseminated, with consequent tuberculous meningitis.

**Differential diagnosis.**—Rheumatism is seldom monarticular, but may occasionally be. Usually, a few days on salicylic acid (aspirin) will cause the pains to disappear. In addition the tuberculin skin tests are negative, and no lesion is seen in an *X ray*. The sedimentation rate is raised in both conditions.

In *pneumococcal* and *gonococcal* joints the differential diagnosis may be very difficult. The advent of the Sulpha drugs however is of real value, as such joints tend to yield to them while tuberculous joints are unaffected. A negative tuberculin skin test is helpful.

*Congenital syphilis* may be excluded by a negative Wassermann. When the knees are affected the disease is bilateral and the swelling painless.

**Treatment.**—Suitable splinting and complete rest are essential. The diet should be full and include all the vitamins. Real and artificial sunshine should be given with deliberate care.

### PSEUDO-COXALGIA

(PERTHES DISEASE)

A rare condition affecting boys more commonly than girls.

**Etiology and pathology.**—A chronic inflammatory condition of the head of the femur usually unilateral but occasionally bilateral. The cause is unknown, it is considered by some to be a low grade staphylococcal infection, but there is no confirmation of this.

**Clinical picture.**—The disease is commonest between five and nine years of age starting with a lump and later giving rise to a pain in the hip. This is in contrast to tuberculous disease of the hip, where pain is the first symptom and lump comes on afterwards. There is limitation of movement of abduction and rotation. The tuberculin test is negative.

*X ray* examination—1 *Early* reveals flattening of the head of the femur  
2 *later* there is fragmentation of the head of the femur and thickening of its neck  
3 *finally* the *X ray* appears almost normal again but the head is inclined to be mushroom shaped (Plate 15).

**Prognosis.**—The vast majority of cases recover almost completely but they remain subject to osteo-arthritis in the hip joint later.

**Differential diagnosis.**—This is from tuberculous disease of the hip see p. 281.

**Treatment.**—Rest at first later caliper splints.

### SYPHILIS OF BONES AND JOINTS

#### 1 EARLY CHANCRE

*Syphilitic epiphysitis* (*Parrot's disease* *Syphilitic pseudo paralysis*)

**Age.**—This condition occurs in the first three or four months of life.

**Pathology.**—The syphilitic infection is at the metaphysis, where softening and separation of the epiphysis may occur. It is common at the upper end of the humerus, and the elbow joint, and is often symmetrical. Periostitis is frequently present also, and may be seen in the *X rays*.

**Clinical picture.**—The affected limb is held perfectly still and is quite flaccid, but not painful.

**Prognosis**—The outlook is good, provided anti-syphilitic treatment is instituted.

**Diagnosis.**—The diagnosis is made certain by the positive Wassermann reaction and the X rays showing the separated epiphysis, 'bitten out' portion of adjacent bone and periostitis in the shaft or other bones.

*Syphilitic dactylitis* occurs in the first two years of life. The swelling of the proximal phalanges is painless and symmetrical, and yields to anti-syphilitic treatment.

## 2 LATE CHANGES

Syphilitic changes in the skull, tibia and synovitis of the knees are present in 39 per cent. of cases of congenital syphilis (Forbes).

**Skull**—These changes occur between the age of five and seven years. The head is 'hot cross bun' shaped and an X ray of the bossing shows both periostitis and osteitis (Parrot's nodes).

**Tibia**—These may be sabre shaped and thickened and the X ray shows osteitis.

*Syphilitic arthritis*—This occurs in older children over the age of six years. There is a bilateral painless swelling of the knees due to a syphilitic synovitis often accompanied by a keratitis (see pp. 348 to 352). This yields rapidly to anti-syphilitic treatment.

## MULTIPLE EXOSTOSES

### (DIAPHYSEAL ACLASIA)

This is more common in males and is a familial and hereditary condition. It is transmitted through the females.

The exostoses arise from bones formed in cartilage (Fairbank) and particularly from the diaphysis near the epiphysis of the long bones especially the knee. There may be many tumours or just one. Clinically the limb may be shortened.

**Treatment**—Complete surgical removal should be carried out if the exostosis is causing inconvenience. Otherwise it should not be interfered with.

## MULTIPLE ENCHONDROMATA (CHONDRODYSPLASIA)

This is familial and is usually found in the bones of the phalanges, carpus and tarsus but also in the radius and ulna. The limb may be shortened. These tumours may soften and may be mistaken for tuberculous dactylitis.

## PRIMARY SARCOMA OF BONE

This condition is rare in childhood. It occurs most frequently in the femur, humerus and clavicle. Prompt surgical interference with X ray or radium treatment, gives a fair proportion of recoveries.

## SECONDARY MALIGNANT TUMOURS

These may be secondary to Wilms' embryoma (see pp. 215-373) or a neuroblastoma, arising from the suprarenal or sympathetic nervous system particularly of the abdomen. In the latter case the tumour metastases are frequently found in the skull and may cause proptosis (Hutchinson type).

## ACUTE SUPPURATIVE ARTHRITIS

This disease may be primary, or secondary to some infective process elsewhere. The organism is most commonly the pneumococcus, but the gonococcus, bacillus of Pfeiffer and bacillus coli have all been described.

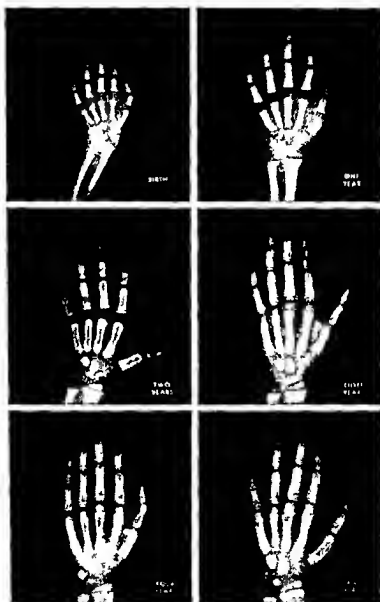


Plate 16.—Radiographs illustrating osseous development from birth to five years of age

(Its courtesy of Messrs Parke Davis & Company)

(See also Plates 18, 19 and 20)



Plate 17.—Radiographs illustrating osseous development from six years to nine years of age (See also Plates 16, 18, and 19)

(By courtesy of Messrs Parke, Davis & Company.)

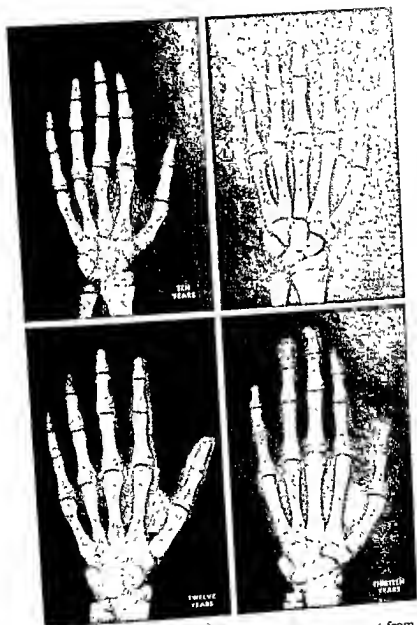


Plate 18.—Radiographs illustrating osseous development from ten to thirteen years of age. (See also Plates 16, 17, and 19.)

(By courtesy of Messrs. Parke, Davis & Company.)

The skin tests of cases of eczema demonstrate allergy to lactalbumin, casein egg white egg yolk cereals fruit juices or other foods. It is suggested that the breast fed infant is reacting to elements in the mother's diet. Infants with eczema tend to have an achlorhydria or hypochlorhydria. One would expect therefore that foreign proteins and breakdown products of the food might gain entrance to the circulation in an imperfectly digested condition.

(2) *Abrasions and friction of the skin causing a local inflammation.* Dermatologists are particularly struck by the traumatic element in eczema.

There is some evidence that the eczematous process is maintained by a low grade secondary infection coupled with constant rubbing and scratching. Probably both elements play a part in each case.

**Clinical picture**—The age of onset is commonly between six months and two years (the dentitional period) but eczema may commence when the infant is only a month or two old. There are two well defined clinical varieties and perhaps the most common is that in which the lesions appear on the cheeks forehead and scalp subsequently affecting any other part. The second variety the flexor type (Besnier's prurigo) is associated with asthma and here the lesions mostly affect the flexures the bends of the elbows and behind the knees. It is not infrequently associated with xeroderma.

In both cases the essential lesions are a characteristic group of papular vesicles. The child rubs or scratches these causing them to burst or weep. This is followed by the formation of a crust. In Besnier's prurigo intense irritation may occur before the visible skin lesions which are often produced by scratching.

If the lesion be extensive the infant is much upset and may be reduced to a pitiful state. In severe cases there is constant rolling of the head and a wriggling movement of the body, feet and hands in an effort

to get relief from the irritation. If the hands are free the child tears at the face and body. Diarrhoea is common and great loss of weight with improvement in the eczema results. Infections of all sorts are easily acquired and badly borne, by these infants.

**Treatment**—It should be explained to the mother at the start that this condition is not infectious and is not due to neglect on her part. The greatest care should be taken that the eczema is not treated so strenuously



Fig. 55 Eczema

disease resembling it most closely is *hæm urticatus* or *urticaria papulosa*. This latter condition is seldom seen between the fingers and toes, however, and is often found scattered over the trunk, especially in the loin.

**Treatment.**—In tiny infants washing gently with soap and water, and applying friar's balsam to all the spots, rapidly removes the infection. Clothing and bedding must be sterilized, and sources of infection, such as the mother or nurse, must be dealt with. In older children the treatment should begin with a warm bath, and a good scrubbing with soap and water. The application of sulphur ointment (B.P.), either full strength for an older child, or diluted with vasoline, equal parts, in younger children, or Sudermo (B.W. & Co.), is recommended. This must be applied all over the body, and re-applied at intervals for three days. During this period the same underclothing or sleeping suit should be used; this becomes impregnated with the ointment and rubs over the skin surface, working the ointment actually into the burrows. On the third day another warm bath should be given and the bedding and clothing should be sterilized; as a rule a cure is soon effected. It may be necessary, however, to repeat this treatment a fortnight later.

Kissmeyer<sup>1</sup> advocates the following mixture. This cured 140 out of 144 children recently, and was successful in 8,000 cases treated in Copenhagen.

Benzyl benzoate	} of each equal parts
Soft soap	
Isopropyl alcohol	

The patient should be thoroughly scrubbed and soaped in a hot bath, after which the lotion is painted on the skin and allowed to dry. A bath the next day, with change of clothing, completes the treatment. Industrial methylated spirit is as effective as isopropyl alcohol.<sup>2</sup>

Where the picture is much complicated by secondary infection cure may be delayed, and treatment for dermatitis or an eczematous or impetiginous condition may have to be undertaken first or simultaneously.

Brain<sup>3</sup> (1913) considers that scabies should be treated, whether complicated by impetigo or eczema or not. He divides treatment into

- (1) the hot bath and thorough soaping,
- (2) the application of an efficient insecticide, the best of which he considers to be emulsion of benzyl benzoate of the National War Formulary (benzyl benzoate, 25 gm., lanette wax S X 2 gm.; water to 100 c.c.), which can be painted on a child one day in the week, and
- (3) the disinfection of clothing and treatment of contacts.

### PEDICULOSIS CAPITIS

This condition was at one time very common, but is now much less so, owing to the fashion of keeping girls' hair short. "Nits" are easily distinguished from scurf or dandruff, which is not fastened to the hair. Infestation leads to scratching, and erythematous rashes may be present

<sup>1</sup> A. Kissmeyer, "Rapid Ambulatory Treatment of Scabies with a Benzoate Lotion," *Lancet*, Jan. 2, 1937, i, 21.

<sup>2</sup> *The Lancet*, Editorial, March 29, 1940, i, 602.

<sup>3</sup> R. T. Brain, "Scabies in Children," *Practitioner*, April, 1913, cl, 251.

is that it is easy to overdose a patient, giving rise to toxic symptoms, with vomiting, headache and albuminuria. With further experience, however, this method will probably prove the most suitable for children.

**Tinea unguum (ringworm of the nails).**—In this manifestation of ringworm the nails are wrinkled pitted and discoloured. Removal of the nails is the surest way of curing the condition. Otherwise it may persist for months or years.

## MICROBIC INJECTIONS

### IMPETIGO CONTAGIOSA

This is due to a streptococcal or staphylococcal infection of the skin. The lesions are various and may appear as simple scabby sores placed on an inflamed base, circinate and imitating ringworm or dry and scaly like seborrhoeic dermatitis or early ulcerative dermatitis.

**Treatment.**—Crusts may be removed by saline compresses. It is usually advantageous to apply antiseptic lotions such as 1 per cent aqueous gentian violet or 1/1000 acriflavine solution. Ointment may be used to soften the scabs to treat septic fissures which occur readily, or for the dry scaly types previously mentioned e.g. an ointment such as hydraz ammon dil and vaseline (equal parts). If the lesions are about the hair, it is best to clip or shave the hair away from the infected parts then foment. Ultra violet light is said to shorten the course of the disease. Strapping the affected parts with zinc oxide elastoplast is beneficial.<sup>1</sup> A course of sulphonamide (see p. 383) is a great help when the infection is widespread.

**Bullous impetigo** is an acute infection of the skin sometimes seen as a wheal about the finger or as *pemphigus neonatorum* in the new born (see p. 67). Penicillin is useful (p. 385).

### FURUNCULOSIS

#### (BOILS)

This disease is due to the *Staphylococcus pyogenes aureus* the infection being in or round a hair follicle. The lesion may take the form of very fine pustules or even of indurated boils. Common sites are the buttocks the perineum, under the arms the back of the neck or the chin. Boils are especially common in children who wet the bed and in school children who get an insufficient number of baths.

**Treatment.**—If the boil is of some days standing the area must be fomented care being taken that the pus is not allowed to drain on the surrounding parts. At intervals the surrounding skin should be wiped over with a weak antiseptic lotion. If the boils are in the perineum or on the buttocks the underclothes should be lined with some smooth material to prevent friction.

**Prevention.**—Treatment for aureus should be instituted. At the very earliest sign of a boil it should be touched with iodine, and then strapped directly (without a dressing) with zinc elastoplast. The plaster should remain on for 10 days or more. Vitamin B and tin preparations



are useful, e.g. —

#### THE CAPSULES

*One or two capsules daily*

- B. Yeast ext. 5 gr  
Colloidal tin and tin oxide 5 gr  
Sulphur præcip ½ gr

760 (Sulphathiazole) is well worth a trial (See p 383) The introduction of penicillin has revolutionized the treatment of staphylococcal infections

#### ERYSIPELAS

This serious infection is not uncommon in infants and young children. It may start from some trivial abrasion or on the other hand it may occasionally follow circumcision, or be found in the skin surrounding the navel or after opening some superficial abscess of the scalp or face. The organism is as a rule, the hemolytic streptococcus. The skin round the site of infection becomes firm, indurated and dark red and there is a very definite edge to the indurated area. It spreads rapidly and may involve the whole of a limb or the head and neck. The temperature is high, the tongue dry and furred and vomiting and diarrhoea are common.

*Treatment*.—Various treatments have been advocated. Much the most efficient lies in a course of one of the Sulpha drugs, particularly sulphadiazine or sulphamethazine. The mortality with these drugs is extremely low (see p 383).

#### SEBORRHEA AND SEBORRHOIC DERMATITIS

In infants, scurf tends to form down the centre of the scalp, especially over the anterior fontanelle.

*Treatment*.—In simple cases the scalp should be anointed with oil and subsequently shampooed with water containing bicarbonate of soda. In some resistant cases, 2 per cent of sulphur and 2 per cent of salicylic acid in a simple ointment should be applied before the shampoo. The daily use of a fine tooth comb will facilitate removal of crusts.

In older children, 'dandruff', due to a mild infection with the bottle bacillus, causes scaliness of the skin of the scalp. This is seen in children over the age of five years, and has probably been contracted from an adult. As it may cause baldness and dermatitis at puberty, it is best treated with frequent shampoos with spirit soap, once or twice weekly.

#### WARTS

(VERRUCE)

Warts are infectious, and have been shown to be due to a filter passing virus.

1. Plain and common warts are found on the hands, feet, and faces of children.

*Treatment*.—Some method of cauterization is most effective. Glacial acetic acid with 1 per cent perchloride of mercury, applied with a match stick, or 10 per cent of salicylic acid in collodion may be used. A ray therapy is often helpful.

2. Plantar warts are more commonly seen in children of boarding-school age. They are highly infectious, and are acquired usually by using the same bathroom mat as an affected child. The warts form on the soles of the feet, on the ball of the foot and the heel and may be mistaken for corns. They are vascular, however, and tender, whereas a corn is hard and avascular.

*Treatment*.—Under general or local anæsthetic the wart should be dissected out completely, or curetted, with subsequent cauterization of the

lase with pure phenol. A recent method described by Thompson is to soak the foot in a 3 per cent formalin solution for 15 minutes daily, for some days, after which the warts are said to fall out of the skin. X ray therapy is also useful for the large solitary wart.

**3 Molluscum contagiosum**—This is also a virus infection and may be acquired at public baths. The papules are small pearly white tumours usually umbilicated and may appear anywhere on the body.

*Treatment*—Curettage and cauterization of the cavity is the best treatment but when the lesions are small erythema doses of ultra violet light may be effective.

### LUPUS VULGARIS (TUBERCULOMES OF THE SKIN)

Lupus is comparatively rare in children. It usually appears on the cheeks and nose and may be due to direct infection from phthisical parents kissing their children, but also arises from a glandular infection, particularly when tuberculous abscesses or glands in the softened stage have been incised. It may appear in any part of the body. At first there is a small tuberculous nodule which gives rise to an indurated patch. This may remain for some months.

*Treatment*—Adams<sup>1</sup> recommends that treatment should consist of either (1) an attempt to remove the disease at one operation under a general anæsthetic by excision or scraping, or (2) a more gradual removal of the lesion by selective caustics or light therapy combined with general treatment. Where only small patches exist excision should be attempted. The caustics he suggests are strong pyrogallol acid ointment 40 per cent in vaseline or aceto-nitrate of mercury. Ultra violet light is of the greatest use and Lomholt's modification of the Finsen lamp is the most efficient apparatus for local treatment.

### HERPES ZOSTER (SHINGLES)

This is due to an infection by an ultra microscopic virus of the posterior root ganglia. It is interesting to note the connexion between chicken pox and herpes. Very often after an adult case of herpes a case of chicken pox follows in the household or vice versa. It is now practically certain that the viruses of herpes zoster and varicella are identical.

*Symptoms*—The eruption consists of a group of vesicles on an inflamed base and is distributed along the sensory fibres of a posterior nerve root. The chest, abdomen, thigh, neck or forehead may be involved. An erythematous rash appears first and becomes vesicular, the vesicles containing clear fluid. Later these dry up and fall off, this stage being accompanied by a slight fever and some malaise and occasionally by neuralgic pains. Scarring does not occur if the scabs are left alone. Herpes of the fifth nerve may be accompanied by corneal ulcer and iritis in which event great damage to the eye occasionally results.

*Pathology*—Lumbar puncture shows lymphocytosis of the cerebro spinal fluid and the posterior root ganglia on section show inflammation and hemorrhage.

*Treatment*—Friction and scratching should be prevented and the lesion should be kept dry with 2 per cent phenol in luteo calamine. Painting with collodion is also useful. A little aspirin may be given at bedtime if the child is restless.

### HERPES FEBRILIS (HERPES PECTUS)

This lesion a crop of small vesicles usually appearing about the mouth or nose is an accompaniment of the common cold. Other sites are the cheeks, the ear, the neck or the fingers. It may accompany pneumonia, cerebro spinal meningitis, tonsillitis or intestinal upsets. The vesicles dry up in a few days and fall off. It tends to recur at the same site.

**Ætiology**—Herpes febrilis has been shown to be due to a virus which is quite different from that which produces herpes zoster

**Treatment**.—Similar to that for herpes zoster

### SYPHILIS OF THE SKIN

Very rarely a syphilitic infant is born with a bullous rash on the palms of the hands and soles of the feet, but such children are usually still born, or die shortly after birth

Bullæ may occur on other parts of the body and are often associated with onychia This variety of congenital syphilis is often called syphilitic pemphigus and is to be distinguished from pemphigus neonatorum The former occurs in very wasted often moribund infants whereas the latter affects the well nourished and otherwise healthy infants and is thought to be a bullous staphylococcal infection of the skin

Usually between the fourth and eighth week a rash appears on the buttocks The skin appears glazed and has a peculiar copper coloured line The rash tends to spread down the legs and up on to the abdomen and may become widespread and extend to the face and trunk The palms of the hands and soles of the feet may have a shiny glazed appearance Condylomata appear about the anus and vulva at the same time

The manifestation of congenital syphilis is likely to be associated with other lesions affecting the periosteum bones and joints as described on pp 348 *et seq*



Fig 57—Rubella like eruption appearing ten days after first administration of M & B 693 Accompanying the rash there is some suffusion of the conjunctivæ and a tendency to confluence of the rash on the extremities normally exposed to sunlight (By order of Dr A R Thompson)

### TOXIC ERUPTIONS ERYTHEMATI

These are rashes due to some toxin circulating in the blood, either a product of defective metabolism, or a food or drug poison or the result of microbic infection (Adanson) These include —

1 The rashes of acute infectious fevers such as scarlet fever, measles, German measles These are dealt with elsewhere

2 Morbilliform and scarlatiniform rashes due to food toxins, sera and enema rashes (toxic erythemata)

3 Erythema nodosum

4 Erythema multiforme

5 Vaccination rashes

6 Certain drug rashes

**Toxic erythemata.**—These rashes resemble those of German measles, ordinary measles, and scarlet fever. The latter may be so closely imitated that the eruption of erythema scarlatiniforme is clinically indistinguishable from a toxic erythema.

There is, however, an absence of fever, sore throat, glandular enlargement and constitutional disturbance in erythema scarlatiniforme, an atypical distribution of the rash supports the diagnosis of toxic eruption.

Among the latest additions to the causes of these rashes are epanutin, employed in the treatment of epilepsy, and the Sulpha drugs in the treatment of acute streptococcal and pneumococcal and other infections.

### ERYTHEMA NODOSUM<sup>1</sup>

Erythema nodosum is a symptom complex and not a disease in itself. It occurs at all ages but is seldom seen before the age of one year.

**Clinical picture.**—Large red blotches or nodes appear on the limbs, especially on the inner surface over the tibia and also but less frequently on the forearm. At the same time the temperature is raised and the child shows lassitude and a poor appetite. The fever may last for two or three weeks but the rash ceases in about 10 days or less, on the average.

**Ætiology and pathology.**—The vast majority of cases are due to a tuberculous sensitization of the skin. Occasionally, the streptococcus is the sensitizing agent, and the author has seen one case due to the meningococcus. Those due to a primary tuberculous infection of the lung and mediastinal glands, or abdominal glands give an exceptionally strong Mantoux reaction. Often a streptococcal throat infection will infect an entire household and all the members will show erythema nodosum, and give an extremely strong streptococcal endotoxin test.

Wallgren<sup>2</sup> believes that streptococcal or other acute infections in tuberculous subjects will produce erythema nodosum more readily, especially in children who have already had erythema nodosum. These post primary cases are of shorter duration in his experience.

**Diagnosis and treatment.**—In every case a very dilute Mantoux tuberculin test say one in ten thou and should be done, and, if positive, a search should be made by X rays for the infecting focus. In cases of streptococcal origin a course of one of the Sulpha drugs should be given. The discomfort caused by the rash may be alleviated by small doses of one of the barbiturates or even aspirin and lead lotion may be applied locally if necessary.

### ERYTHEMA MULTIFORME

An erythematous eruption characterized by symmetrical distribution on the limbs and face and by the exudative nature of the erythema (Adamson).

**Ætiology and pathology.**—Occasionally swelling of the joints and pain suggest a connection with rheumatic fever, but there is probably a closer relationship to allergy and serum disease. There is evidence of cell proliferation, however and it is possible that it is microbic in origin, probably streptococcal.

<sup>1</sup> W. R. F. Collis, "Erythema Nodosum," *Brit. Med. Jour.*, Dec. 23 1933, II, 116. Harold Emsberg "Erythema Nodosum and Tuberculosis," *Amer. Jour. Dis. Child.*, Dec. 1933 XLVI, 1797.

<sup>2</sup> Arvid Wallgren, "Post-Primary Erythema Nodosum Tuberculosis," *Acta Tuberculosa Scandinavica*, Vol. 10 fasc. 4.

**Symptoms.**—The lesion appears on the backs of the hands and forearms, sides of the face and neck, and on the legs. There are disc-like dark red erythematous patches, usually somewhat raised. There may be vesicles or bullæ at their summits. The central portion may fade producing a ring-like appearance, resembling a pupil and iris. This variety—*erythema iris*—is very liable to frequent relapse.

**Course.**—(Adamson) The condition lasts from a few days to a week, and then fades, but it may recur over and over again. A rise of temperature is observed at each eruption, and the eruption may appear in the mouth.

**Treatment.**—The patient should remain in bed and be given salicylates internally, with calamine lotion or lead lotion to the affected parts. If a streptococcal focus is located a Sulpha drug should be given.

## URTICARIA

(LICHEN URTICATUS URTICARIA PAPULOSA HEAT SPOTS NETTLE RASH HIVES)

**Ætiology.**—The cause of this affection is not entirely understood but it appears to be bound up with the following factors:—

1 *General overfeeding*—The spots although they may appear throughout the year, are very much more common in the summer months. It is in the warm weather that children are most liable to overfeeding. The great majority of those affected by urticaria are overweight or inclined to be rather plump, though it does occasionally occur in the thin child and it should be taken as a sign that the digestion is being taxed to the limit of its powers.

2 The overfeeding may be on *heating foods* such as porridge, gruels, yolk of eggs, cream or much butter.

3 Strawberries, cherries, plums, raspberries, gooseberries, bitter oranges or lemons may all play a part in bringing about an attack in some children.

4 Shell fish, rabbit, ducks, eggs, meat juices may also produce these lesions in some susceptible children.

**Clinical picture**—The spots may be typical of nettlerash with a slightly raised red area which irritates. On the other hand they may resemble the bite of an insect. Common sites are the backs of the hands, the forearms, legs, loins and buttocks, but they may appear on almost any part of the body except the scalp. The common type appears as small, shotty, hard, white blisters which when scratched contain clear fluid and give place to a small raised, inflamed or red area. These may be mistaken for early chicken pox. Both types are known as "heat spots," "nettle rash," "hives," or "itching spots." If the tops are torn off the spots may become infected.

**Treatment**—A careful review of the child's diet should be made to ensure that he is not being overfed. The best course is to adopt the principle that the less often he is offered food the less likely he is to be overfed. Three meals a day should be given after the age of one year. If the weather is warm, heating foods should be stopped, and groats, cream, butter, eggs and milk should be limited. Cod liver oil and malt is often the offender. Raw fruit, especially very acid raw fruit, should be stopped for the time being. Tangerines are a good substitute for oranges, as they

are less acid. Shell fish, raw meat, juice and ducks' eggs must be avoided. The bowels should be kept well open with grey powder at bedtime and milk of magnesia the following morning. The spots should be dabbed with calamine lotion to allay the irritation.

The following prescription will be found useful —

Dilute hydrochloric acid	10 minims
Syrup	30 minims
Water to	60 minims

One teaspoonful in a little orange juice three times daily with meals.

Those cases due to group 4 (alkalyl) will benefit by this treatment.

### DRUG RASHES

Most drugs and indeed most of the common foods may in idiosyncratic subjects produce an eruption. The rashes are like those described under toxic erythemata but the abrupt onset and absence of any constitutional disturbances may give a clue to the diagnosis. A few of the common drug eruptions are given as examples of these rashes.

**Bromide rash.**—Bromide is often supplied in the form of gripe water or soothing powders, or the mother may be taking the drug for some purpose and excreting it in her milk. The rash appears as vesicles or pustules on the face, body and limbs and later these become hard raised crusta varying from the size of a pea to that of an almond. In some cases the eruption is suggestive of smallpox. When the crusta fall off they leave no scar and usually the lesions disappear rapidly with the cessation of the drug but occasionally a further slow resolution takes place.

**Belladonna rash.**—This is scarlatiniform and is accompanied by dilated pupils, dryness of the mouth, sometimes vomiting rapidly and excitement.

**Sulphonamide rash.**—After the administration of any of the sulphonamide group the patient may show some cyanosis. This is due to sulphæmoglobinæmia. This is not a serious symptom and the drug need not be discontinued. Eggs and tomatoes in the diet apparently do not aggravate the cyanosis. Rashes occur in 6 per cent. of cases (C. H. Brown, 1939). They are purpuric, scarlatiniform, morbilliform or maculopapular and the distribution may be general or patchy. Occasionally they are itchy. The rashes are toxic in origin and disappear rapidly when the drug is withdrawn. Eruptions are quite often provoked by local sulphonamide applications and an intractable eczema or dermatitis may result.

**Luminal** and other barbiturates may produce a blotchy erythema particularly over the joints in susceptible individuals. Quite often the eruption simulates German measles.

**Epanutin** may produce a morbilliform rash and temperature but the glands are not swollen. This subsides with its withdrawal.

**Dermatitis venenata**—Many individuals react to external poisons in plants and in aromatic oils or other applications e.g. poison ivy (*Rhus toxicodendron*) and the primrose (*Primula obconica*). The hands, faces or legs may come in contact with the plant and a severe dermatitis result. There may be a simple redness and swelling but often large bullæ or vesicles are formed, and irritation is marked. The illness lasts from one to three weeks.

**Treatment**—If there is much constitutional upset the child is best put to bed, and sedatives, such as the barbiturates, should be given. A local

application of bicarbonate of soda, or washing with strongly alkaline soap, may lessen the spread. Such applications as lead lotion should be used freely, or liquid paraffin, to which has been added either 2 grains of menthol to the ounce, or 5 grains of carbolic acid (Griffiths)

Individuals who are susceptible may be desensitized yearly. For treatment a toxin (antigen) is also available (Griffiths)

### SERUM RASH (SERUM SICKNESS)

A rash may follow injections of antidiphtheritic, antistreptococcal, antitetanic or other sera. As a rule, it appears about ten days after the injection, but it may come earlier or later. It is often accompanied by an urticarial rash, which may be blotchy and erythematous, resembling measles. There is tenderness in the joints, a temperature malaise and occasionally actual vomiting; these symptoms persisting perhaps for two or three days or longer. The joint pains with the subsequent rise of temperature have been known to last as long as six weeks. Calcium gluconate, 20 gr., or small repeated doses of chloral should be tried. Ephedrine,  $\frac{1}{4}$  gr., twice or three times daily, is indicated, or 1-8 minims of 1-1,000 solution of adrenaline should be given subcutaneously. For the skin irritation use menthol, 60 grains, paraffinum album molle 1 oz.

### ENEMA RASH

The cause of this rash is not clearly understood. It is bright red, and appears on the knees, elbows, buttocks, face and chin. It may be a blotchy erythema or actually scarlatiniform. Most frequently it occurs within 12 hours of the administration of the enema, but may appear almost immediately.

### VACCINATION ERUPTIONS

**Local.**—There may be superadded infection at the site of vaccination and erysipelas may result. This is very rare.

**Generalized erythema.**—This is toxic, and resembles a serum or enema rash in its distribution. Usually, it appears seven or eight days after vaccination. It is, however, very rare indeed.

A generalized vaccinia which occurs 7 to 11 days after the primary vaccination is a sparse eruption of umbilicated papular pustules resembling smallpox. Auto infection from the primary vaccination lesion may result in an acute secondary vaccinal eruption which is very severe and the skin may be markedly oedematous.

### PSORIASIS

This is a recurring eruption with a hereditary or familial history in about 10 per cent. of cases. It has a characteristic appearance and is rare before the age of five or six years. The causation is unknown.

**Clinical picture.**—A child in good health develops either an extensive eruption like an exanthem, usually on the body, or the eruption may be limited to the extensor aspects of the limbs, especially the elbows and knees. The scalp and nails may also be affected. The lesions consist of small pale scaly, pink papules. They start as tiny points or patches, and extend to patches an inch or more in diameter. Adamson says the following points are characteristic —

- (1) The circumscribed patches grow by extension at the margin, not by coalescence of smaller patches.
- (2) They remain always dry.
- (3) On scraping the scales become more obvious until at length a level is reached where there are no more scales but a dry, red smooth surface, which bleeds on further scraping.
- (4) The red patch which remains after the scales have been removed shows no infiltration and fades on firm pressure, that is, the lesion is a scaly macule not a papule.
- (5) On the scalp the patches are usually thicker and more scaly.

**Pathology**—(Adamson) Psoriasis shows a dilatation of the vessels of the papillary layer with enlargement and prolongation of the papillæ, and a catarrhal inflammation of the epidermis resulting in œdema and imperfect cornification of the horny cells so that these adhere to form scales instead of exfoliating normally.

**Treatment**—Dieting and general treatment does not seem of value.

**Local treatment**—A very useful ointment is—

*℞* picis carb 80 minims

*℞* g hydrarg ammon dil to one ounce

Those resisting this treatment should be rubbed with ung dithranol (Cignolin 1/1000). Alternatively ung chrysocolum (B.P.) should be used but care should be taken that it does not get near the eyes. An improvement may be expected in about a fortnight.

### PITYRIASIS ROSEA

Pityriasis rosea is uncommon in children. The characteristic features are—

- (1) It commences as a solitary patch called the herald patch. This may appear on the chest, neck, abdomen, arm or thigh.
- (2) A week or ten days later similar patches in great number appear over the body, particularly the trunk.
- (3) The patches are the size of a finger nail and show three zones, a central yellowish zone of finely wrinkled epidermis, outside this a narrow scaly band and a fine smooth pink margin. The patches are irritating.
- (4) The eruption lasts about six weeks and constitutional disturbances are slight or absent.

**Treatment.**—After a daily warm bath salicylate ointment (15 grains to the ounce) is well rubbed in.

### ALOPECIA

(BALDNESS)

Baldness may occur over a wide area due to depression of the general health of the child by some febrile illness. Thus after measles, scarlet fever or typhoid, the hair may come out, but with improvement in the general health a fresh growth occurs. Baldness may also occur about an inflamed area of the scalp, such as a boil or impetiginous patch.

**Alopecia areata**—The characteristic of this form of baldness is that it occurs in patches, and may be mistaken for ringworm. It may extend to



permanent baldness. Constitutional factors—toxic, nervous or endocrine—probably form the ætiological basis.

**Diagnosis**—The bald patch is usually smooth shiny and devoid of scales—unlike that of ringworm. A close examination of the stumps will show that they resemble an exclamation mark (!) thick above the skin but becoming narrow as the hair reaches the root. The eyebrows may also be affected.

**Prognosis**—Recovery in children usually occurs in six to eighteen months (Adamson). Where alopecia is universal the prognosis is not so good.

**Treatment** *General*—Consideration should be given to the general health and elimination of focal sepsis. The psychological aspects of the child's life should be carefully reviewed and adjusted if necessary. General tonics, ultra violet light (local and general) and a change of environment are most beneficial.

*Local*—Some mild irritant to the scalp should be tried to improve the local blood supply. Any cantharides lotion is suitable.

### SCLEREMA NEONATORUM

This rare condition is sometimes seen in newly born infants, or in infants who have had a severely debilitating illness such as gastro enteritis. Firm brawny lard like lumps may be felt on the thighs shoulders and back. The skin is firmly adherent to the patches, which are occasionally purplish red.

**Pathology**—An examination of the fat in such areas shows the presence of a low melting point fat only and there may be crystal formation in the fat cells with a foreign body giant cell and granulomatous reaction to it.

**Prognosis and treatment**—In the author's experience the majority of such cases recover. If the general health can be improved the lumps gradually disappear.

### SCLERODERMA

This condition is identical with that in adults and is very rare in children. The affected areas are indurated and have a yellowish waxy surface. In older children the induration may be associated with hardening of the muscles. It has certain features suggestive of sclerodermata myositis.

### VITILIGO (LEUCODERMA)

Children with this condition show patches of skin from which the pigment has disappeared. They are most noticeable in the summer months, when the surrounding skin is pigmented. As a rule they are symmetrically distributed such as on both knees or a patch on both shoulder blades. The health of the child is unaffected. The cause is unknown.

**Treatment**—No specific treatment is known but the pale areas may be toned to match the surrounding skin with walnut juice or tea. Occasionally, good results have followed painting the white areas with 10 per cent solution of oil of bergamot in spirit, with subsequent exposure to ultra violet light.

### SEBORRHOEA OLEOSA

This is a condition of hypersecretion of sebum, the subjects of which have large sebaceous glands especially about the sides of the nose, and central parts of the face. Seborrhoeic subjects are also very liable to acne, boils, eczema and infections of the mucous membranes. This appears to be a permanent condition, and an inborn characteristic rather than a disease, treatment is only palliative e.g., frequent use of soap and hot water, and sulphur lotions. X rays to the face are of benefit.

## PITYRIASIS CAPITIS

Scurf is probably due to a mild infection of the scalp with the organism called *pitryosporon*. In susceptible subjects the infection may produce either a seborrhoeic dermatitis or an eczematous process which spreads from the scalp to the face and flexures. It is important therefore to treat the scurfy scalp. 2 per cent. of sulphur and salicylic acid in coconut oil is a suitable application and frequent shampoos should be given until the scalp is clear.

## ACNE VULGARIS

Acne vulgaris occurs in those who suffer from seborrhoea oleosa, and is due to an infection of the sebaceous glands with the acne bacillus.

**Ætiology.**—Between puberty and the age of about 18 it is usual for the sebaceous glands to hypertrophy, and acne is common therefore at this age but it may also appear in young children or even in infants if greasy preparations are constantly applied to the skin. Both males and females are affected. The sebaceous glands become blocked because of inflammation, and blackheads are formed. These in turn may become further infected, and become small pustules. It is worse at the menstrual periods in the female.

**Clinical picture.**—The spots appear on the forehead, cheeks, sides of the nose, temples, chin, chest, back of the neck and between the shoulder blades.

**Treatment.**—Since acne may leave permanent scarring and disfigurement and produces an inferiority complex in some individuals, the treatment is important.

**General treatment.**—Great attention should be paid to the bowels, and fresh air and exercise are essential. If there is anaemia, this should be treated with iron and other tonics. General ultra violet light is very useful.

**Local treatment.**—The sheet anchor is soap and hot water to remove the excessive grease from the skin and scalp. A lotion should be applied in some form such as 2 per cent. of sulphur in calamine lotion. Roxburgh suggests —

Zinc sulphurata	22 grains
Pot sulphurata	20 grains
Acetone	120 minims
Aqua rose	1 ounce

This should be applied with the hand two or three times daily, after washing and should be allowed to dry on the skin.

Once daily use the following cream —

Ung. acid. salicyl. (2%)	1 ounce
Glycer. amyl.	1 ounce
Aqua ros. geran., q.s.	

Ultra violet light and especially  $\lambda$  rays, will be found of real value. Quarter pastille (100 $\gamma$ ) doses of  $\lambda$  rays, from three to five doses in all, at weekly intervals, are recommended by Roxburgh.

## CHAPTER XVI

### INFECTIOUS DISEASES <sup>1</sup>

INFECTION disease plays an extremely important part among the diseases of childhood and assumes special importance during the child's school life. In former times the children of even the well to do had some if not most, of the infectious diseases before school age. Now as a result of immunization, care of contacts, observance of quarantine and among other factors, better ventilation and hygiene children may reach the secondary or public school having had only one or two infections. This has introduced a new problem into school life among older children. There is no doubt in the author's mind that putting off infectious disease until a child is over five and stronger is a very great advantage. The mortality of most infectious fevers is much higher among children of the pre-school age than among those above it. This is borne out in the following mortality statistics<sup>2</sup> —

	Under 5 years	Mortality 5-15 years	TOTAL under 15 yrs
Measles	388	60	448
Scarlet fever	33	48	79
Whooping cough	788	10	798
Diphtheria	730	900	1630

An excellent summary of the position in regard to infection and the period of quarantine necessary in various diseases is given in a publication issued by the Medical Officers of Schools Association<sup>3</sup>

#### SCARLET FEVER

**Incubation period**—This is from 1 to 7 days, but usually 2-4 days. A child who has had scarlet fever should be isolated for 4 weeks (28 days), and may mix with others at the end of this period provided there are no complications e.g., discharging nose or ears.

**Mode of infection**.—This is either by droplet spread from some infected individual or carrier to a healthy person, or by direct contact with infectious discharges, or through milk. It is said that it can be transmitted on some infected article of clothing or in a book (fomites), but this view is open to question.

<sup>1</sup> Report of Committee on Immunization, including Vaccination, *Brit. Med. Jour.*, June 22, 1935

<sup>2</sup> J. D. Robertson and G. W. Ronaldson. "Acute Infectious Fevers" (Heinemann), 1940

<sup>3</sup> 1912 Registrar-General's Figures (England and Wales).

<sup>4</sup> "A Code of Rules for the Prevention of Communicable Diseases in Schools, Issued by the Medical Officers of Schools Assoc., 10th Ed. (J. & A. Churchill), 1940.

**TABLE XX**  
**INCIDENCE AND MORTALITY OF SCARLET FEVER**

	1936		1937		1940		1941		1942	
No. of notifications (all ages)	104 623		95 631		46 951		50 797		41 104	
No. of deaths										
Under 15 years	339		4 1		106		98		9	
Over 15	100		75		48		30		25	
Total No. of deaths	439		249		154		133		104	
Percentage of notifications	0.432		0.265		0.332		0.261		0.251	
	No.		No.		No.		No.		No.	
	per cent.		per cent.		per cent.		per cent.		per cent.	
Under 1 year	16	3.23	9	2.6	9	2.9	5	3.76	6	5.7
1 to 5 years	123	29.9	143	41.9	43	9.2	5	4.8	7	9.0
5 to 10 years	24	2.3	9	2.36	45	7.9	28	11.0	34	2.7
10 to 15 years	45	4.09	50	6.6	11	1.14	8	6.01	12	11.7
Total on per 10 years	339	75.5	2 4	75.56	106	68.91	98	75.5	9	5.9
No. over 15 years	106	11.48	70	21.34	48	31.14	35	26.43	20	24.1
Grand Totals	439	100	249	100.00	154	100	133	100	104	100

**Seasonal incidence**—Scarlet fever is most prevalent in the late summer and early autumn but may occur in epidemic form at any time of the year.

**Age incidence**—It is uncommon in infants under one year, and most common between 5 and 9 years. Sporadic cases where no infective source can be traced are frequent and it must be concluded that there are a large number of scarlet fever carriers.

**Ætiology**—Recent work has shown that scarlet fever is due to a hæmolytic streptococcus which produces an erythogenic toxin. The throat is the usual site of attack but infection of wounds by the same organism produces surgical scarlet fever. Numerous types of streptococci are capable of causing scarlet fever.

**Diagnosis**—The diagnosis may sometimes be extremely difficult as it depends on the rash which can be simulated by enema rash by toxæmia by food poisoning or by Sulpha drug rashes. The sore throat can also be simulated but cultivation from the throat in scarlet fever patients shows a hæmolytic streptococcus. The Dick test is always positive in the first few days, but changes to negative in the second or third week. Peeling a late sign, is pathognomonic of scarlet fever but occurs less frequently in patients who are given adequate doses of serum.

The Schultz Charlton test, which is performed by injecting intradermally 0.2 c.c. of a 1 in 10 dilution of scarlatinal antistreptococcus horse serum, is an excellent means of diagnosis. The serum is injected into the skin of patients exhibiting a rash and where the rash is due to scarlet fever there is a blanching about the point of injection 1 to 10 hours later, which persists for from 12 to 72 hours.

**Symptoms and clinical picture.**—In very mild cases, which are often called *scarlatina*, nothing may be seen beyond a very slight soreness

of the throat and a rise of temperature, with a transient rash, the whole illness subsiding in 3 or 4 days and ending in complete recovery. In the more severe cases there is an abrupt onset, usually with vomiting, and both headache and intense sore throat. The child is ill and dull. The pulse and respiration rate are increased, the skin is hot, the temperature considerably raised and the cheeks are flushed. The rash appears in 24 to 48 hours but occasionally not until the third or fourth day. In some cases, however, it occurs first of all. There is usually only one vomit.

**The rash**—This is erythematous and comes out on the trunk and limbs (except the palms of the hands and soles of the feet), and on the neck towards the lower borders of the jaw, where it merges with the flush on the face. Round about the mouth there is a pale area, the so called 'circum oral pallor'. The rash may last for a few hours or as long as a week.

**Desquamation**—At the end of a week or ten days the skin begins to peel, starting round the neck and upper part of the chest then generally the skin of the hands and feet peels last, the tags of skin can be seen for some time afterwards at the roots of the nails. The peeling is generally in proportion to the intensity of the rash and lasts from 4 to 8 weeks. It is not a sign of infectivity.

**The tongue**—The so called "strawberry tongue" can be seen on the first or second day—a white tongue with red papillae showing through. As the disease progresses, the tongue becomes red all over, the throat being painful and the tonsils enlarged and fiery red with enlarged glands at the angle of the jaw. The younger the child, the greater the glandular enlargement.

**Complications**.—The commonest are otitis media and cervical adenitis which are seen in about 3 per cent of cases. Glomerulo nephritis is found in 1.5 per cent of cases. Both these complications should be dealt with as suggested on pp 188 and 209. Rheumatic carditis or arthritis is found in approximately 1 per cent of cases. Some of the rarer complications are ulcerative stomatitis, lobar pneumonia and infantile hemiplegia.

Relapse, due to re-infection of children with another and probably more potent type of streptococcus, is common and can be prevented by nursing children separately or in wards where only one type is present.

**Prognosis**.—In fatal cases, where the infection has been especially virulent, death is due to a general septicæmia, with an accompanying myocarditis, or to some complication such as pneumonia or nephritis. Generally speaking, the prognosis for life is excellent, whereas for ages under two the mortality is about 15 per cent, for ages over two it is less than 1 per cent. Haemorrhagic rashes are a bad prognostic sign.

**Treatment**.—The patient is nursed in bed from the beginning. The diet should be the fever diet described on p 329. If the temperature is unduly high, above 103°, tepid sponging should be resorted to, with water at about 90° F. A little aspirin (2½ grains), or sodium salicylate (5 grains) and potassium chlorate (3 grains) is extremely useful. The throat may be swabbed with weak iodine or perchloride of iron or perchloride of mercury. The administration of quantities of alkalis is specially indicated, and from 30 to 60 grams of bicarbonate or citrate of soda should

be given daily during the febrile period. Bland drinks, including much water, should be insisted on. The greatest possible care should be taken of the mouth, to prevent stomatitis, and it should be swabbed every two or three hours during the day with some mild antiseptic such as glycolyl nioline peroxide of hydrogen, or permanganate of potash. If there is any suggestion of involvement of the heart, the patient should be kept lying perfectly flat for a long period. A careful examination of the urine for albumin should be made daily. A watch must be kept on the ears for any signs of otitis media. The child should be kept in bed until the temperature has been normal for one week, and the pulse-rate is normal.

**Serum**—Scarlet fever antitoxin may be given as a prophylactic. In most cases 2½–5 c cm is sufficient to protect 'contacts' but 0.5–1 c cm of protein digested serum will have the same effect as 5–10 c cm of concentrated scarlet fever antitoxin. As treatment, 2.5 to 7.5 c cm of the serum should be given. Opinion appears to vary on the efficacy of the Sulpha drugs in conjunction with serum. Some writers claim that they lessen the incidence of complications while others claim that the incidence is not affected.

**The Dick test**—This is performed by injecting intradermally 0.2 c cm of diluted filtrate obtained from a broth culture of the streptococcus of scarlet fever which has been found to cause erythema 1 cm in diameter when injected into susceptible subjects. This is called the skin test dose. Heated filtrate is used as a control to be certain that the patient is not reacting to the horse serum. If the patient is susceptible to scarlet fever a reddened area appears in from 6 to 24 hours round about the injection. This test is of considerable value as the 'susceptibles' can be immunized in an epidemic.

**Active immunization**—This is done by injecting scarlet fever toxin at fortnightly intervals. An approximate dosage for children of 5 to 10 years is 500, 2,000, 5,000, 10,000, 20,000 skin test doses. Double this dose is used for adults. Reactions are fairly common and only those running risks of exposure should be immunized. The immunity lasts for years, compared with the two or three weeks immunity conferred by passive immunization with serum.

## RUBELLA

(GERMAN MEASLES)

**Age-incidence.**—This disease may attack children at any age, but it is more common after five. It is spread by droplet infection from child to child. Rubella is not among the most common infectious diseases although it was widespread in 1940.

**Incubation period**—This is from 10 to 21 days, but is usually 15 or 16 days, the child being infectious 1–4 days before the appearance of the rash or other symptoms. He may return to school when 20 clear days have elapsed from the last date of exposure to infection, but if he has been himself infected he cannot return until 7 days after the appearance of the rash.

**Clinical picture and symptoms.**—The disease commences with very slight constitutional symptoms, the temperature rising as the rash

comes out. There may be coryza and sore throat, and there is usually enlargement of the glands behind the sternomastoid and round the occipital and mastoid region. The axillary and inguinal glands often share in the glandular enlargement. As a rule, however, the rash is the first indication. Occasionally conjunctivitis (pink eye) accompanies the disease. There are few or no complications.

**The rash**—The distribution of the rash is on the face and scalp, trunk and extremities. It may be morbilliform or a diffuse erythema, not usually as general as that of scarlet fever. Occasionally, it is urticarial. It rarely lasts longer than three days and may have completely disappeared in 24 hours. As it tends to be mealy, the disease is usually mistaken for measles, but, on the other hand, it may be mistaken for urticaria or nettle-rash, due to some toxæmia or drug. In measles, unlike rubella, there is some indisposition and coryza two or three days before the appearance of the rash. Glandular fever is sometimes mistaken for rubella and vice versa.

**Treatment**—The child should be kept in bed until the temperature has become normal and should be isolated for one week after the appearance of the rash. No other treatment is necessary.

The mortality is negligible. Second attacks occur, but one attack confers immunity, probably in 90 per cent.

## MEASLES

(Morbilli)

**Incubation period**—This is from 7 to 21 days, usually 10 days, the child being infectious at least 4 days before the appearance of the rash or other symptoms. Convalescent serum prolongs the incubation period. A child may return to school when 16 clear days have elapsed from the last date of exposure to infection, but if he has himself been infected, he cannot return until 14 days after the appearance of the rash. The causal agent is a virus. One attack confers permanent immunity.

**Clinical picture**—Four to five days before the rash appears, the temperature gradually rises to 102°–101° F and remains there while the rash is present. Then, after three or four days, the rash fades and the temperature falls. During this febrile stage, the eyes and nose run, and the face is puffy. The child looks heavy and appears to have a bad cold. The inside of the mouth and cheeks shows Koplik's spots, and laryngitis or croup may be present. The rash appears behind the ears and round the mouth, and then spreads rapidly over the rest of the body. The throat is sore, and a troublesome cough may develop with catarrhal bronchitis. The temperature remains up from one to five days or longer. There is conjunctivitis, so that the eyes are sore and tend to discharge. Photophobia may occur without conjunctivitis. The glands of the neck, particularly those behind the sternomastoid, become enlarged and tender.

**Koplik's spots**—These appear on the first, second or third day of the prodromal period, and are best seen inside the cheeks, especially towards the posterior part of the mouth. They are small, about the size of a pin head, raised, and bluish white, surrounded by a red areola. Their absence, unfortunately, does not in any way exclude a diagnosis of measles.

**Rash**—The rash of measles is characteristic. It starts as small pink macules which rapidly form larger, irregular shaped patches or blotches. Large areas may become involved giving an erythematous appearance. This rash may last from 3 to 5 days, and often leaves a faint brown, stained appearance behind it. The temperature falls with the falling of the rash. Prodromal rashes occur during the period of incubation. They are usually blotchy and erythematous.

Vomiting and diarrhoea frequently accompany the rash but are easily controlled with small doses of nepoche.

**Complications**—The most common are pneumonia, otitis media, conjunctivitis and gastro-enteritis. An extreme rarity is measles encephalomyelitis<sup>1</sup> the mortality from which is high. The illness may begin with

TABLE XVI  
INCIDENCE AND MORTALITY OF MEASLES<sup>2</sup>

	1940	1941	1942	
No. of notifications (all ages)	410,270	306,962	273,594	
No. of deaths Under 15 years	823	1091	458	
	1919			
Over 15 years (for the two years)	210		10	
Total No. of deaths for the two years	2129		458	
Under 1 year	No. 249	No. 324	No. 138	% 29.7
1 to 5 years	478	626	252	54.8
5-10 years	69	124	64	11.8
10-15 years	12	17	6	1.4
I.e. Total under 15 years	828	1091	458	97.6
No. over 15 years			10	2.2
Totals			478	100

convulsions along with the rash and fever, but it is more usual for it to commence four to six days after the appearance of the rash, the author has seen a case in which drowsiness and other symptoms came on a fortnight after the rash. Some cases make a quick recovery, others show permanent signs of an upper motor neurone lesion. Transverse myelitis sometimes occurs. Repeated lumbar puncture and convalescent measles serum subcutaneously are indicated.

**Treatment**—The child should be put to bed in a darkened room, the eyes being thus rested. The diet should be the fever diet (see p. 329).

<sup>1</sup> Parnes, Blake et al., "Encephalitis in Measles," *Brit. Med. Jour.*, March 20 1927 1, 687.

<sup>2</sup> Registrar-General (England and Wales).



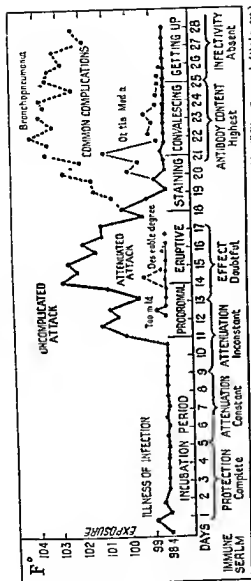


Fig. 58.—The effect of serum on the course of the attack in the London Measles Epidemic 1935-36. The dosage of serum and the time table of its administration are as follows:

Nature of serum	Dose in c.c.m.	Dosage factor	Route	Prophylaxis		Treatment
				Prophyl.	Attenuation	
Convalescent	8-10	Age x 2	Intramusc.	1st-2nd day	2nd-3rd day	Within 8 days of onset 2 dosage factor ages x 4 Route intravenous.
Normal adult	10-10	Age x 4	Intramusc.	1st-3rd day	3rd-4th day	
Whole blood	Ex. able ill serum.	Age x 4	Intramusc.	1st-3rd day	3rd-4th day	
Lacertal extract	7.0-10	Age x 5	Intramusc.	1st-3rd day	3rd-4th day	

with an abundance of bland fluids—cereals and other carbohydrates should be given when possible. The bowels must be kept well open and some alkaline saline such as milk of magnesia or Eno's Fruit Salts should be given. A mixture of sodium salicylate (5 grains) and potassium chlorate (3 grains) may be given three times daily, or

Aspirin in powder	5 grains
Milk sugar	3 grains
Three times daily	

The child should be kept in bed until the temperature has been normal for from two to four days. The greatest care should be taken of the mouth—swabbing it out two or three times daily with peroxide of hydrogen, glycothymoline or glycerin and borax or an aqueous solution (0.5–1 per cent) of gentian violet.

The treatment by serum from a convalescent patient has not been very satisfactory and in our present state of knowledge is not advisable. As a prophylactic, however, there is no doubt that serum given to contacts as early as possible after the third day of the incubation period either completely aborts or greatly attenuates the disease.

The disease could not be more graphically described than in the accompanying diagram (Fig. 62).

**Passive immunity**—Serum from patients convalescent from measles or from adults known to be immune may be used for prevention or for attenuation of the disease in contacts.<sup>1</sup>

*Dosage for prevention*, in children 1 to 5 days after exposure 5 c.c.m. of convalescent or 10 c.c.m. of adult immune serum.

*Dosage for attenuation* in children 1 to 5 days after exposure 2.5 c.c.m. of convalescent or 5 c.c.m. of adult immune serum and double the dose for children inoculated 6–9 days after exposure.

Whole blood (20 to 40 c.c.m.) and placental extracts are other passive prophylactic agents.

A course of one of the Sulpha drugs (*see p. 383* for dosage) is said to lessen complications.

## MUMPS<sup>2</sup>

### (EPIDEMIC PAROTITIS)

Mumps is an infection of the salivary glands, usually affecting the parotids bilaterally. It may occur at any age but is most common between the ages of 5 and 15 and is conveyed by droplet spread from one child to another. The disease is due to a filter passing virus which is present in the patient's saliva.

**Incubation period**—This is from 14 to 28 days, but is on the average 18 days, and the child may be infectious 1 day before the appearance of the symptoms. He may return to school when 28 clear days have elapsed from the last date of exposure to infection but if he has been infected himself, he must not return until 7 days after all swelling has disappeared and not earlier than 14 days from the beginning.

<sup>1</sup> Code of Rules for the Prevention of Communicable Diseases in schools, 10th Ed., Churchill 1949.

<sup>2</sup> W. Gunn *Lancet* April 2nd 1928, 1739.

**Incubation period.**—This is usually about a week, but may vary from 5 to 14 days. Whooping cough is infectious as soon as the first cough appears. Contacts may return to school when 21 clear days have elapsed from the last date of exposure to infection, but an infected child may not return for 5 weeks from the beginning, or until whooping has ceased for at least 14 days, or until cough plates have shown him to be bacteriologically negative.

TABLE XXII MORTALITY (ENGLAND AND WALES) REGISTRAR GENERAL

Year	Total under 15 years	Under 1 year		1 to 5 years		5 to 10 years		10-15 years	
			per cent.		per cent.		per cent.		per cent.
1940	67	257	53.2	993	47.6	35	4.9		—
1941	255	1718	52.2	1682	46.0	65	2.8	5	0.07
1942	798	487	61.0	301	2.9	10	1.1	—	—

**Bacteriology and pathology.**—It is generally agreed that whooping cough is due to the Gram negative Bordet Gengou bacillus (*Hæmophilus pertussis*) which is found in the cough droplets early in the disease. The infection is essentially a laryngeal and tracheitis, much semi-purulent material mixed with mucus being produced. The cause of the whoop is not known, but it may be due to a secondary enlargement of the tracheal glands pressing on the trachea or larger bronchi. A help in the diagnosis may be found in the blood-count where a distinct lymphocytosis occurs. The count may rise from 20 000 to 80 000 per c. mm. with a proportion of 60 or 70 per cent. of lymphocytes. In the author's opinion the blood count is seldom helpful and definite enough in those cases where the diagnosis would be of most value. If the child coughs spontaneously on to media composed of defibrinated blood mixed with an equal quantity of 3 per cent. agar containing a little extract of potato and glycerine the organism can be cultivated in over 90 per cent. of cases in the early days of the disease. For diagnostic purposes in doubtful cases such cough plates are recommended. The organism cannot be recovered usually after the third week although symptoms may still be present. These release tests save much time in children of school age.<sup>1</sup>

**Onset and clinical picture.**—Generally speaking, whooping cough may be divided into three phases: (1) the catarrhal, (2) the paroxysmal and (3) the phase of recovery. In a classical case these three stages last approximately a fortnight each, but there is very great variation.

1 *The catarrhal phase.*—The disease begins like a severe cold, and there is catarrh of all the upper respiratory mucous membranes. There is a tendency to slight fever, and a persistent hacking cough gradually becomes worse especially at night. The nose runs, the face is puffy, and a little albumin may appear in the urine. A blood count shows an increase of the leucocytes to 15 000 or 30 000, a high proportion being lymphocytes. Gradually, the cough becomes more and more spasmodic, i.e., it occurs in sudden severe spasms, followed by a comparatively long period quite free of cough. The child is awakened or disturbed more and more frequently

during the night. A small ulcer may appear on the tongue, where the frenum impinges on the lower incisors. This is most frequently seen in children under two years of age. The duration of this phase varies considerably, in some cases being as short as 4 or 5 days.

2 *The paroxysmal phase*—At about this time or just before, whooping cough is strongly suspected or has been diagnosed, as the child begins to whoop and vomit. Young children, who seem able to anticipate an attack of coughing, run to their mother or nurse for support. Along with the bout of coughing the last food taken may be mechanically vomited. Each paroxysm of coughing ends in such a spasm that on inspiration a long drawn out whoop may be heard, the face becoming cyanosed and the eyes bloodshot. A typical paroxysm may be said to consist of a series of short expiratory coughs, with a long inspiratory whoop which continues until a plug of mucus is dislodged, when the spasm ceases. Mucus and saliva stream from the nose and mouth during the attack. This phase generally lasts about a fortnight but in some cases may be almost, or entirely, absent or last for many weeks.

3 *The phase of recovery*—During the last fortnight, the whole process rapidly retrogresses. The whoops become less frequent at night, so that the child's sleep is less disturbed. During the day there is less vomiting, and consequently the child begins to gain weight. By the end of the fortnight, in the average case, the cough has practically ceased.

*Recurrences*—In some cases there is a continuance of the whoop, and bouts of coughing occur for many months. There seems no explanation, except that the child has acquired a habit or trick of whooping. Other children, who seem to recover completely, develop a slight pulmonary catarrh or bronchitis some months later, with a return of what appears to be a typical whoop.

*Complications*.—Undoubtedly one of the most distressing complications is convulsions. At the end of a bout of coughing the child, now extremely cyanosed, passes rapidly into a convulsion, which continues for some minutes. Such cases on the whole do extremely badly, and there appears to be a tendency to minute cerebral hemorrhages at this time. Certainly, cases of hemiplegia are not uncommon after whooping cough convulsions. In one case seen recently by the author, the child was left physically normal but mentally most unstable and excitable, a picture that might be expected to follow encephalitis.

*Epistaxis* is another quite common complication. It should be treated by keeping the child in the upright position, and by applying cold compresses.

*Wasting*, due to vomiting and accompanying anorexia is also a severe and common complication which may become a real problem in certain cases. It is advisable during the stage when vomiting is most frequent either to feed the child again immediately after the vomit or to give frequent small feeds of thin fluids containing glucose, which can rapidly pass out of the stomach.

Miliary tuberculosis, ingrafted on or following whooping cough, may occur, and should be suspected where the whooping-cough fails to subside, or where the pulmonary complications are severe.

**Broncho-pneumonia** is probably the most serious and frequent complication of whooping cough. In these circumstances the term secondary pneumonia is employed. There is a tendency for such pneumonias to remain unresolved leading to pulmonary fibrosis or bronchiectasis. Emphysema may also develop.

**Treatment**—1 *General management*—In the catarrhal phase the child is often best kept in bed, or at least off its legs. This is even more essential in the paroxysmal phase of the severe type of case as, with much whooping and vomiting the child suffers from loss of weight and exhaustion. In mild cases if the child is afebrile, he should be up and about and in the garden if the weather is suitable.

**Medicinal**—There is no satisfactory medicinal treatment. A mixture containing tincture of belladonna 5 minims, and potassium bromide 3 grains may be given from 4 to 6 times daily. As a sedative, chloralhydrate in doses of from 2 to 4 grains may be useful, or occasional doses of one of the phenol-arbitone preparations, say,  $\frac{1}{2}$  to  $\frac{1}{2}$  a grain twice or three times daily (see p. 397).

**Chemotherapy**—None of the Sulpha drugs appears to have any effect on the organism of pertussis but complications are said to be lessened or can be treated by these drugs in the usual way.

**Tonics**—In the later stages of the disease cod liver oil and malt, or cod liver oil emulsion or halibut oil preparations are indicated.

**Vaccines and convalescent serum**—There is no convincing evidence that these are of any use, once the disease has commenced.

**Diet**—The diet should be light and essentially non-irritating to the pharynx, by this is meant the avoidance of dry or crumbly foods such as toast or biscuits. Milky foods such as junkets, milk puddings, custards, cereals well soaked in milk, gravy and potato and nutritious soups, are best. It may be necessary to discontinue the regular times of meals for a short time, and feed young children immediately after a bout of whooping and vomiting.

**Prophylaxis**—*Vaccine*—The efficiency of prophylaxis vaccination varies enormously and it is safe to say that most vaccines are useless. Apparently, the important point is the size of the dose, and whether the freshly isolated organisms are of the "smooth" or "type 1" phase. The dose should be large up to 80 billions, given in 3 to 4 injections at fortnightly intervals. The vaccine prepared by Sauer appears to have given good results. Alum precipitated toxoid pertussis has been used, but is still in the experimental stage though it has given encouraging results.

2 *Convalescent serum*—Contacts of whooping cough can be protected, or the disease much modified by 15 to 20 ccm of convalescent serum or double this quantity of adult whole blood. The earlier it is given the more effective the protection.

**Convalescence**—Whooping-cough is a debilitating disease and in addition to tonics a change of air is most beneficial.

\* Donald Paterson and J. M. Smith. "The Value of Vaccines in the Treatment of Whooping-cough." *Brit. Med. Jour.*, May 6 1933 1 713.  
D. Paterson, R. H. Bailey and J. P. G. Walker. "Control of Whooping-cough with Serum and Vaccine." *Lancet*, Aug. 17 1935 II 501.  
L. V. Sauer, *Jour. Amer. Med. Assoc.* 1933 XI 1443.  
1 June 1935 *Acta Paediatrica* 1935 IV 1.

## CHICKEN-POX

(VARICELLA)

**Ætiology.**—Chicken pox is caused by a filter passing virus

**Incubation period.**—This is from 12 to 20 days, the child being infectious for two days before the vesicles appear. A child may return to school when 20 clear days have elapsed from the last date of exposure to infection but, if he has been infected himself, he must wait until all the scabs have fallen off, particularly those on the scalp.

**Symptoms and clinical picture.**—Chicken pox is one of the mildest diseases of childhood, but may occasionally take a very severe form. As a rule, the first signs of infection are the vesicles, but the child may have been out of sorts for a day or two previously. The rash appears as a series of red papules, which rapidly become vesicles or blisters. The temperature is usually  $100^{\circ}$ – $102^{\circ}$ .

**Distribution of rash.**—The rash appears first on the trunk, face and scalp, then on the limbs, sometimes in the mouth. It is moderately itchy.

Fever develops with the appearance of the rash but the temperature falls to normal within a day or two. Successive crops of the eruption may appear for several days, and pocks may be seen alongside one another in different stages of development.

**Complications.**—Among the commonest complications are tonsillitis, laryngitis and tracheitis. The vesicles however may become infected and abscesses result.

**Treatment.**—The child should be kept in bed until the temperature has been normal and the naso-pharyngitis quiet for three or four days. The main treatment should be directed towards the prevention of scratching in order that scarring (particularly of the face) does not occur and the lesions do not become infected. This is best done by wearing loose soft smooth clothing by the application of dusting powder or calamine lotion and by keeping the child very cool. In girls the vesicles on the face may be pruned with collodion which tends to prevent scarring. In small children, wearing gloves or splinting the arms may be necessary to prevent scratching.

## DIPHTHERIA

**Ætiology.**—Diphtheria is caused by infection with the Klebs Loeffler bacillus.

**Incidence.** *Age*—It may occur at any age. It is rarely seen before one year, because immunity is acquired from the mother. The period of greatest susceptibility is from 1 to 10 years of age.

*Season*—It is most common in the autumn and winter months.

*Routes*—Toys, clothing, bedding, pencils and books may remain contagious for a long period especially if they are kept in the dark (Roller's opinion).

**Spread of infection.**—It is chiefly spread by healthy or sick carriers, droplet infection disseminates the disease by coughing, sneezing, spitting, kissing or speaking. Very rarely, contaminated milk is a cause of spread but it does not act as a source of infection to human beings.

**Pathology**—The organism tends to remain localized at the site of infection and does not invade the tissues deeply the toxins being absorbed by the blood stream and lymphatics. The faucial membrane consists of fibrin masses of necrotic cells and bacteria. The heart shows acute parenchymatous degeneration the kidneys and liver cloudy swelling and fatty degeneration. The suprarenals show very little change. Nerves in the region of the lesions show degeneration of the medullary sheath. The blood shows a slight leucocytosis.

**Symptoms and clinical picture**—The three chief forms of diphtheria are faucial, laryngeal and nasal which may occur separately or in combination. Very occasionally the ear, anus, vulva or skin abrasions may be infected.

TABLE XXIII  
INCIDENCE AND MORTALITY OF DIPHTHERIA  
(Registrar General, England and Wales)

	1936		1937		1938		1939		1940	
Total notifications (all ages)	58,20		61,435		48,281		50,727		41,404	
No. of deaths										
Under 15 years	584		770		2,506		2,300		1,650	
Over 15	497		193		180		291		191	
Total No. of deaths	3,081		2,963		2,400		2,441		2,186	
	No.	per cent.	No.	per cent.	No.	per cent.	No.	per cent.	No.	per cent.
Under 1 year	8	2.68	81	2.73	38	2.33	75	2.63	43	2.2
1 to 5 years	1,34	23.54	1,031	21.82	618	26.87	1,106	21.80	767	25.45
5 to 10 years	1,426	18.77	1,350	24.11	1,043	22.05	980	21.14	718	29.5
10 to 15 years	24	11.12	346	11.74	84	11.45	279	8.67	279	9.80
Total deaths under 15 years	2,544	83.59	2,706	93.56	2,400	92.70	2,300	90.50	1,638	80.35
Deaths over 15 years	497	6.41	193	6.30	180	7.30	291	9.50	191	10.45
Totals	3,081	100	2,963	100	2,480	100	2,441	100	2,186	100

**FAUCIAL DIPHTHERIA** Incubation period—One to seven days most commonly two to five days.

**Onset**—This is insidious and although older children may complain of a sore throat may not be suspected in the young child who complains of general malaise, anorexia and pains in the limbs. Vomiting may occur.

**Appearance of the throat**—In the early stages one or more patches of membrane resembling old ivory or wasleather appear on the tonsils rapidly spreading to the uvula. The fauces are red and cedematous. The membrane is firmly attached to the subjacent mucosa and attempts to remove it cause bleeding.

**Glands**—The cervical glands are enlarged and may be much enlarged resulting in the so-called bull neck. The odour from the throat is fetid and characteristic and there is often a blood stained nasal discharge.

**Temperature**—This is usually only slightly raised or may be normal throughout.

**Facial appearance.**—Although the face may be flushed in mild cases in those of any severity with much toxæmia the complexion is pallid and grey.

**HÆMORRHAGIC DIPHTHERIA**, with bleeding from the mucous membranes, occasionally occurs and carries with it a high mortality

**LARYNGEAL DIPHTHERIA** The first signs of the onset of this are a change in the character of the voice. Later the breathing becomes more difficult, with indrawing of the suprasternal space and recession of the ribs, and over action of the muscles of respiration

**NASAL DIPHTHERIA** This is usually an extension of the faucial form, but may occur by itself, in which case it is comparatively benign. In the isolated nasal form the lesion is in the anterior portion of the nares and may be accompanied by general symptoms, such as headache, shivering and vomiting, or with no symptoms apart from a profuse, watery or purulent blood stained discharge. The cervical glands may be enlarged

**Complications.**—(a) *Paralysis* (see p. 246)

(b) *Cardio vascular system*—Various cardiac arrhythmias are found. There may be simple sinus arrhythmia, which is of no significance. Extra systoles, paroxysmal tachycardia, auricular fibrillation, auricular flutter, and complete or incomplete heart block. Most significance is to be attached to changes in the heart sounds in the first fortnight of the disease, especially in severe cases (Rolleston). Where cardiac disease is suspected, an examination by an electrocardiograph is necessary

**Second attacks.**—About 2 per cent of cases get second attacks. It should be clear, therefore, that an attack of diphtheria does not necessarily render a child immune

**Diagnosis.**—The clinical diagnosis is of much more importance—since on it treatment must be instituted—than the bacteriological diagnosis, which will not be available for 24 hours. A small dose of antitoxin given on the first day of the disease is of more value to a child than a large dose given 24 hours later (Rolleston). The clinical diagnosis is made on the appearance of the membrane covering the tonsils, uvula and soft palate, together with enlarged glands, nasal discharge, *faux*, and the fact that the membrane cannot be separated from its bed without causing bleeding

**Differential diagnosis.** (1) *Faucial diphtheria*—The chief conditions likely to be mistaken for faucial diphtheria are acute follicular tonsillitis, Vincent's angina, acute leukaemia, glandular fever, streptococcal cervical adenitis and mumps. (For differential points applying to each of these see their descriptions)

(2) *Laryngeal diphtheria*—This should be differentiated from simple laryngitis or croup and laryngo-tracheo-bronchitis. (For differential points applying to each of these see their descriptions)

(3) *Nasal diphtheria*—Acute rhinitis and foreign body in the nostril. (For differential points applying to each of these see their descriptions)

**Prognosis.**—This depends on (1) the age of the child, generally speaking the younger the child the higher the mortality, (2) the day of the disease on which antitoxin is given, generally speaking, the mortality rises steeply for each day the antitoxin is delayed, (3) the type of infection, and severity of reaction to the infecting organism, e.g., where the infecting



organism is of the "gravis" type there is a correspondingly serious prognosis. Some children react badly to diphtheria, and from the start show palatal oedema, profuse nasal discharge, much glandular enlargement, oral fœtor and hæmorrhages into the skin and, despite big doses of antitoxin, these fail to clear up.

(4) Other signs of bad prognostic significance are cardiac failure, shown by weakening of the first sound at the apex, thready pulse, galloping rhythm, persistent vomiting, progressive enlargement of the liver and early palatal paralysis, particularly in association with paralysis of the diaphragm and pharyngeal muscles.

**The Schick test.**—This test makes it possible to determine whether an individual is susceptible or immune to diphtheria. It is performed by injecting 0.2 c.c. of a diluted diphtheria toxin into the skin of the fore arm, intracutaneously. A control of heated toxin is used on the opposite arm. If the patient is not immune to diphtheria, a reaction appears round the injection, indicating that he is susceptible and should therefore be actively immunized. If the result is negative, nothing further need be done.

**Prophylaxis and immunization against diphtheria.**<sup>1</sup>—Most infants carry over from their mother some immunity to the disease for the first few months but rapidly lose this towards the end of the first year. It is customary to immunize infants or young children against the disease, and it appears that once this is done properly they are immune for life. There are a variety of preparations open to the practitioner. (1) *Toxin antitoxin mixture (T & M)*, the immunizing efficiency is low (70 per cent), with a high reaction; it is given in three doses of 1 c.c., at weekly or fortnightly intervals. (2) *Toxoid anti-toxin floccules (T & F)*, this has a very high immunizing efficiency, with a very low liability to reaction, and the dosage is as for T & M. (3) *Formol-Toxoid (F & T)* also has a very high immunizing efficiency, but there may be some reaction, the dose is as for T & M. (4) *Alum Precipitated Toxoid (A.P.T.)*, this has a very high immunizing efficiency, and very little reaction generally, although there may be some local reaction in the arm. It is best given in two doses. The first dose would be 0.2 c.c., followed four weeks later by 0.4 c.c. In children who are allergic to various sera, Parke, Davis & Co. make Toxoid antitoxin (goat serum) three doses. Three months after the injections, the child should have a Schick test done to make certain that immunity has been gained.

Communities and schools properly immunized can be almost free from diphtheria.

**Diphtheria in the inoculated.**—A small proportion of those inoculated develop diphtheria when exposed to infection, it is usually of a mild type and seldom proves fatal.

**Treatment.**—Rolleston states that "as a general rule, the size of the dose should be determined by the extent and character of the membrane in the throat, the presence or absence of faucial and palatal oedema, the concomitant adenopathy and fœtor, and the date of the disease."

<sup>1</sup> H. J. Parke, Immunization and the Diphtheria with Alum Precipitated Toxoid (A.P.T.), *Trav. Med. Jour.* Feb. 1926, 1, 113.

1 Dosage of antitoxin — A general guide for dosage of diphtheria antitoxin is —

- 1 A simple nasal infection — to 10 000 units, intramuscularly
- 2 A moderate faucial infection 10 000 to 30 000 units intramuscularly
- 3 A severe naso-pharyngeal infection 50 000 to 100 000 units partly intravenously and partly intramuscularly
- 4 A severe faucial naso-pharyngeal infection 60 000 to 100 000 units intravenously
- 5 A laryngeal infection 90 000 to 30 000 units intramuscularly
- 6 A faucial laryngeal infection 20 000 to 50 000 units, intramuscularly or intravenously

Injections are best given intramuscularly into the outer surface of the thigh. In severe cases the warmed serum may be given intravenously into the median basilic or internal saphenous vein directly or by the drip method.

2 General treatment — The patient should be kept in bed for 8 weeks in mild cases and 8 to 12 weeks in severe cases. He should be recumbent for the greater part of this time but may have one pillow at the end of 14 days, pillows may be gradually added until he is sitting upright or he may get up for a few moments at the end of seven weeks. If no cardiac abnormality or diaphragmatic paresis is present he may be up by the 8th week.

3 Circulatory failure — If vomiting begins rectal or intravenous saline and glucose drips should be used. coramine may be injected in collapse. The foot of the bed is best raised on blocks.

4 Laryngeal diphtheria — In severe cases with much dyspnoea tracheotomy should be performed, in the less distressed cases intubation or laryngeal suction may be instituted.

5 Paralysis — (See post diphtheritic paralysis p 246)

Freedom from infection — Before release from quarantine cultures of the throat, nose and of any discharges should be taken. two successive negative results are a fair indication of freedom from infection.

## TYPHOID FEVER

### (ENTERIC FEVER)

**Etiology** — This disease is caused by the *Bacillus typhosus* a Gram negative motile organism, which is killed by boiling or by carbolic acid in a strength of 1 in 20.

**Incubation period** — This is commonly ten to fifteen days but may be from five to twenty three days.

**Mode of infection** — As a rule the patient is infected by means of water, milk or milk products such as ice cream or cream tarts by shellfish or oyster meat or fish or by direct contact with a typhoid patient or carrier. Outbreaks in institutions have occasionally been traced to a carrier in the kitchen. Ice and salads have both been found infected the latter from contaminated soil.

**Clinical picture and diagnosis** — Generally the onset of the attack is gradual with a slight headache, epistaxis and staircase like rise of temperature. In a moderately severe case there is a gradual onset of delirium and drowsiness, followed by extreme prostration and toxæmia. The disease may be ushered in by acute diarrhoea and vomiting. The period of continuous fever may vary from a week or 10 days to 6 or 10 weeks. In average case in childhood lasts 3 to 4 weeks. There is almost invariably some intestinal disturbance either diarrhoea or vomiting at the beginning or later on some degree of looseness of the bowels with pale watery stools. Occasionally however there is obstinate constipation. Gradually in the third or fourth week, the

temperature settles, the tongue becomes clean, the appetite improves, the delirium ceases and the patient begins to recover.

**The spleen**—This is found enlarged in the vast majority of cases during the first few days of the illness, a sign which should always be sought.

**The blood**—The white count shows a normal number of white cells or a leucopenia, e.g., 5,000 white cells per cmm., 60 per cent being polymorphonuclears and the remainder lymphocytes.

**Agglutination (Widal) test**—This is seldom positive before the tenth day and reaches its maximum about the sixteenth day. It should not be considered positive unless it agglutinates the typhoid organism in a dilution of 1 in 25. As a rule, the test gives a positive agglutination in a dilution of from 1 in 250 to 1 in 1,000.

**Cultures of the blood and urine** may be positive during the first 4 or 5 days of the illness.

**Pulse rate.**—In a classical case of typhoid the pulse is slow and bounding, but this cannot be relied upon.

**Rash**—The so-called "rose spots" are diagnostic and appear at the beginning of the second week, usually over the abdomen, chest and back. They are round and are about the size of split peas or smaller and disappear on pressure. They are few in number, usually not more than 4 or 6 being present at a time.

**Headache**—This is, as a rule, very severe and, coupled with opistaxis, is most suggestive.

**Wasting**—One of the most alarming features of typhoid fever is the tendency to wasting. A limited quantity of food only can be administered and digested, whereas with a high continuous temperature, restlessness and delirium, the caloric requirements of the patient are extraordinarily high, consequently severe wasting almost invariably occurs.

**Pathology.**—During the first three or four days typhoid fever is a blood infection and there is an actual septicaemia. It is possible during this stage to recover the organism from the blood and urine. After this time the infection appears to settle in the bowel, especially in the lymphoid tissues, causing hyperplasia and ulceration of the Peyer's patches and swelling of the mesenteric glands and spleen. Evidence of toxicity is shown by cloudy swelling throughout all the organs and there may be a severe peripheral neuritis or encephalitis. The Peyer's patches slough and separate, leaving shallow ulcers, which tend to perforate. The sloughs may show a tendency to hemorrhage.

**Prognosis.**—In children the mortality is extremely low, perforation being almost entirely unknown and hemorrhages exceedingly rare. J. D. Rolleston in "Acute Infectious Fevers" gives the mortality under five years as 8 per cent., from five to ten years about 6 per cent., and from ten to fifteen years 7 per cent. This rate is much higher than in a series of cases seen by the author.

**Treatment.**—For the first few days, as a rule, the case is not diagnosed. Probably, during this time, in view of the gradually rising temperature, drowsiness, and vomiting or diarrhoea, the usual treatment for acute gastro-enteritis has been instituted. The child will have been given a dose of castor oil and put on a very light diet. Once the diagnosis is made, however, the nursing and treatment should be systematized along the following lines.

The patient should be given abundance of sugar water or fruit juice and water, and fed regularly on diluted milk thickened with some starchy preparation, such as Benger's Food, groats, cornflour arrowroot, or broth thickened with potato. If curds appear in the stool, the milk should be peptonized. As large a quantity of the starchy milk food should be given as the patient is able to digest. When the temperature begins to subside, more and more solid food should be administered, although it is at this time that the sloughs begin to separate and there is fear of perforation. Despite this, potato and gravy, a little raw steak, and eggs in various forms should be given. If there should be any tendency to constipation, some liquid paraffin preparation should be employed to keep the motion soft and prevent damage or straining on the weakened bowel.

The nursing is of the utmost importance. During the acute stages, seed drinks or tepid sponging may help to prevent hyperpyrexia. A little aspirin is also of value. The greatest possible care should be taken of the mouth in order to prevent ulcer-formation, and several times daily it should be swabbed out with peroxide of hydrogen, glycothymoline, glycerin of borax, or a 1 per cent aqueous gentian violet solution.

A water bed is desirable to prevent bed sores, and all the pressure points should be sponged over daily with some hardening lotion such as methylated spirits or alcohol. All excreta should be collected and dealt with by disinfecting with 1 in 20 phenol. Where hemorrhage is suspected in view of rapidity of the pulse, blanching and collapse absolute rest should be imposed with an ice bag on the abdomen. Where perforation is suspected on account of a sudden rise of temperature with abdominal pain and tenderness and obstruction, laparotomy should be undertaken at once. The patient should be kept carefully under observation in view of the possibility of laparotomy.

**Serum therapy**—In epidemics both at Bournemouth and Croydon antityphoid serum was used with some success, and it appears rational to give it a trial in most toxic cases.

**Complications**—*Bronchitis* is so commonly associated with typhoid that it may be looked upon as part of the disease and may call for treatment by one of the Sulpha drugs.

*Myocarditis* is met in severe cases. The heart becomes dilated, the pulse is thin and thready, the colour is poor and the heart's action weak. It may be necessary to administer coramine or digitals (see pp. 167-197) and nasal oxygen or an oxygen tent will sometimes tide a patient over this phase. Rolleston states that myocarditis is present in 4/5 of all cases of typhoid.

**Rare complications in children**—Perforation of the bowel and hæmorrhage from the bowel have already been mentioned. Osteomyelitis and arthritis are rare, the former occurring most often in the head of the tibia. Polyneuritis may also occur.

**Prophylaxis**—The vaccine usually consists of 1 000 million *Bacillus typhosus* and 500 million each of *Bacilli paratyphosus A* and *C* per 1 c.c.m.<sup>1</sup> A child may be actively immunised by two doses of this vaccine given at intervals of 10 to 12 days. The dose for adults is 1 c.c.m. followed by 1 c.c.m. The dose for children is one half the adult dose for older children and one fourth the adult dose for younger children. There is a difference of opinion among authorities on active immunisation during an epidemic. H. Schutz<sup>2</sup> working at the Lister Institute, claims that experimentally there is no danger in this practice, and that it is of real value.

### PARATYPHOID FEVER

This is much more prevalent in London than typhoid fever and most of the epidemics of recent years have been of this variety. Paratyphoid has a clinical picture like that of a very mild form of typhoid. The duration of the fever is shorter, being from two to three weeks at most (fig. 59). The feeling of malaise and general upset may be severe, but is usually mild or even slight. The organisms responsible are the *Bacillus paratyphosus A* and *B* and these can be demonstrated on culture of the blood or urine, or later the positive agglutination (Widal) test is given, thus confirming the diagnosis. The prognosis and treatment are the same as in typhoid fever.

### UNDULANT FEVER<sup>3</sup>

This infection which is becoming more readily recognized throughout the British Isles is caused by *Brucella melitensis* and *Brucella abortus*. All cases seen by the author, and most of those reported point to cow's milk as the source of infection.

**Age and sex incidence**—It is much commoner in males than in females. The average age of the author's cases was 7 years.

**Clinical course**—The onset is preceded by a feeling of malaise, and the symptoms of the common cold. The temperature fluctuates sometimes being up continuously but at other times subsiding. It may rise suddenly to a peak and then come down over a period of days then stay normal for a few days, and once more undulate. This slight fever may continue for many months. In one of the author's cases it continued for a year. Sweating especially in the second half of the day, is a feature. The spleen is palpable in a good proportion of cases. Constipation is the rule rather than diarrhoea.

**Diagnosis**—A positive blood agglutination confirms the diagnosis. The intradermal test may be of value. Abortin or Brucellin being used as antigen.

In the differential diagnosis, stress should be laid on the long continued fever,

<sup>1</sup> H. Rehnke, *Lancet* 1932 II, 612. H. C. M. Williams, Inoculation against the typhoid group of ferres, *Proc.*, June 1931, vol. 25.

<sup>2</sup> H. Weldon, *Undulant-Fever Clumps*, etc. "Undulant Fever," *Lancet* Dec. 28, 1935 II, 1112. "Reports on Public Health and Medical Subjects," No. 59 (H.M. Stationery Office, 1935).

<sup>3</sup> D. Paterson and C. Hardwick, Undulant Fever in Children, *Arch. Dis. Child.*, 1935, vol. 63.

the feeling of prostration in the afternoon, the marked sweating and the palpable spleen. Such conditions as typhoid, paratyphoid and tuberculosis must be excluded.

**Prognosis**—All the author's cases recovered.

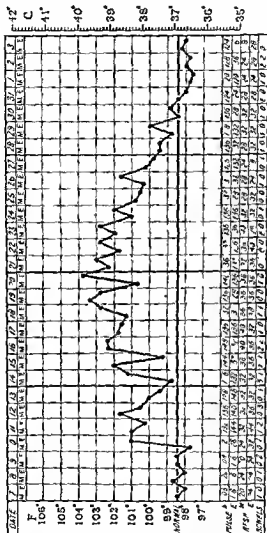


Fig. 59.—Complete temperature chart of a case of paratyphoid in a child aged 5. Note that the pulse-rate ranges from 108 to 145, and that at no time did the child have diarrhea.

**Treatment.**—There is no specific treatment. Vaccines, protein shock, and antipyretics have all been used with varying success, but the disease appears to run its course. Lately it has been claimed that sulphaiazine is more efficient in treating this condition than any other of the Sulpha drugs which have been tried from time to time. Chemotherapy has not proved a success in the author's hands.

### INFLUENZA

Influenza may be either of the epidemic type, in which event a large proportion of the population is affected, or sporadic. In the great pandemic

of 1918 the proportion of infants and children affected was extremely small. The infection may show such a great variety of clinical features that the diagnosis is always of the greatest importance.

**Ætiology and pathology.**—Smith, Andrews and Laidlaw<sup>1</sup> have shown that influenza is due to a *filter-passing virus* which can be obtained in nasal washings from the patient. The incubation period is from a few hours to three days. The chief damage to the body appears to be toxic and cloudy swelling of all the organs is found at autopsy. An examination of a case of pneumonia complicating this disease will reveal a number of organisms, including the streptococcus, pneumococcus and Pfeiffer's bacillus.

**Clinical picture.**—This may show great variations both with the particular epidemic and from year to year. Usually the onset is acute, the temperature rising suddenly to 102° or 103°, with aching all over, a disinclination for food and a tendency to vomit. There may be a slight erythema or sore throat, and the tongue is dry and coated. Mild cases subside in 2 or 3 days, but the more severe cases may last for a week or longer. In addition to a general infection there is also, it is believed, a septicæmia. Each epidemic may attack some particular system and there is a preponderance of symptoms in that part of the body. Thus the epidemic of 1929, besides the usual general symptoms of influenza, brought with it a strong tendency to *otitis media* and its complications. The *respiratory system* may be specially affected with mild or severe catarrh of the upper respiratory tract, in some cases extending down to the smaller tubes, giving rise to bronchitis and broncho-pneumonia. *Gastric influenza* may commence suddenly with profuse vomiting and diarrhoea accompanied by abdominal pain. In very young infants the disease may rapidly prove fatal, but in older children the symptoms subside in 2 or 3 days. *Nervous symptoms* may complicate influenza and the connexion between this disease and encephalitis is not yet fully explained.

**Difficulties in diagnosis.**—During an epidemic the diagnosis is extremely simple, especially when other members of the family are infected. The chief difficulty, however, arises in sporadic infections, and the name "influenza" is often given without justification because the symptoms are so indefinite. Later the true nature of the complaint is disclosed and the mistake becomes apparent. The diagnosis of influenza should not be made until every other possibility has been excluded, and then only with the very greatest misgiving. The blood count will be a great help, as influenza shares with typhoid fever the peculiarity that there is no leucocytosis, but rather a tendency to leucopenia.

**Prognosis.**—On the whole the prognosis in children is excellent, the mortality being chiefly among those with pulmonary complications.

**Treatment.**—The child should, where possible, be kept apart from other members of the family, with a maximum supply of fresh air. The temperature of the room should be maintained at 60° to 65°. Where catarrh of the upper respiratory tract is severe a cresol or vapour lamp may be used for one or two hours daily. In mild cases, half a tablet

<sup>1</sup> W. Smith, C. H. Andrews and P. F. Laidlaw *Brit Jour Exp Path.* Dec 16 1933, xvi, 56r

of aspirin (2½ grains) should be given three times daily to a child of five or six.

**Chemotherapy**—In more severe cases sulphadiazine or sulphathiazine should be given from the start. Although it is clear that the Sulpha drugs do not have any effect on the virus they appear to control the accompanying infection, i.e., the streptococci and pneumococci, which so frequently invade the sinuses, ears and lungs (see p 383).

**Diet**—Frequent drinks of sugar water or fruit juice and water, well sweetened, should be given the child being encouraged by keeping the drinks at hand. When a desire for food has returned, starchy preparations such as groats Cream of Rice or potato and gravy should be given at meal times the milk which should be diluted, being limited to half or three quarters of a pint in the day. Once the tongue has begun to clear up a more solid and generous diet should be resumed at once. Where the temperature is high tepid sponging with water at about 80° F is useful.

**Bowels**—The bowels should be kept open. With infants this can be accomplished by giving milk of magnesia a teaspoonful every hour until the bowels move. Where this is difficult a saline enema of 3 or 4 ounces should be given repeatedly until a free action has taken place. In older children grey powder, 3 to 5 grains, should be given at bed time followed the next morning by two or three teaspoons of milk of magnesia an hour before breakfast. A rhubarb and soda mixture (see pp 102 339) is also useful, particularly when the tongue is coated.

Where the symptoms are largely gastro intestinal, infants will require the therapy described on p 105 to make good their dehydration. In older children bland fluids with a slow return to full diet, are all that is necessary, except in the most severe cases, where intravenous saline and glucose may be required.

**Complications**—Complications such as otitis media should be dealt with as described on p 139. For pneumonia the treatment described on p 148 should be instituted.

It is the author's experience that some complication should be suspected if the temperature remains above 100° or 101° after the third day. A careful examination of the urine should be made in all cases.

TABLE XXIV

THE REGISTRAR GENERAL'S FIGURES FOR 1940-1942 (ENGLAND AND WALES)  
DEATHS FROM INFLUENZA

Year	Totals	Under 1 year	1-5 years	5-10 years	10-15 years
1940	760	259	298	57	47
1941	502	169	147	57	29
1942	311	156	106	28	21

### VACCINATION<sup>1</sup>

Vaccination remains the chief method of prophylaxis against smallpox, and children who have been vaccinated are almost entirely immune to the disease. It is best done in infancy, best of all in the first few months of life.

<sup>1</sup> C. J. McSweeney "The Clinical and Epidemiological Characteristics of Variola Minor and its Differential Diagnosis" *Quart Jour Med* No. 96 July 1951 xxiv, 457

It has been shown that complications such as encephalitis only occur in older children who are being vaccinated for the first time. Complications and mortality from vaccination in infancy are nil.

**Method**—The arm should be carefully cleansed with ether or ether soap and the calf lymph applied at one or at most two spots. Then with a sterile needle or scalpel tiny scratches should be made just through the epidermis, insufficient to draw blood. Immediately after this a sterile dressing should be applied and firmly strapped in place with adhesive plaster. In girls the leg preferably the outer surface of the calf should be used, if the buttocks are chosen the site of vaccination may become infected. The following directions should be given to the mother.

- 1 The arm must be kept absolutely dry. If it should get wet in bathing the dressing must be changed at once. Do not use boracic lint or powder.
- 2 Do not remove the pad or look at the arm for three days provided it does not seem too tightly fastened or uncomfortable.
- 3 In three days, begin undoing the dressing and looking for vesicles. Once they begin to appear, the dressing must be changed every day. In this way the dressing is not allowed to stick to the lobe and no difficulty is experienced in changing it.
- 4 If the child is very restless it may be necessary to fasten the dressing with adhesive tape.

5 Once the vaccination "takes" it reaches its height by the seventh day and then commences to heal rapidly.

By the third day the vaccination should begin to take beginning as a red area. By the fifth or sixth day a vesicle is present. By the seventh day the maximum inflammation has taken place and from this point the whole thing subsides rapidly. When vaccination is done under aseptic conditions the reaction is trivial.

An alternative method of vaccination is by the intradermal route. Calf lymph is injected intradermally into the skin of the arm or leg. The smallest amount possible is injected with a syringe and needle.

#### POST VACCINAL ENCEPHALITIS

Encephalitis is a rare complication and the nervous symptoms arise from 9 to 15 days after vaccination. It may start with drowsiness, vomiting and convulsions. Squint may be absent, trismus is often present. The complication occurs most commonly in older children, not in the infant and young child and it does not occur in those who have been previously vaccinated. After a short illness the child recovers completely without sequelae or after-effects. This is the only serious complication of vaccination and may be easily avoided by vaccination in infancy. The mortality appears to be about 30 per cent.

The morbid anatomy somewhat resembles that of encephalitis lethargica (see p. 26).

#### Fever Diet

(IN INFLUENZA, ACUTE INFECTIOUS FEVERS, OTITIS MEDIA, MASTOID DISEASE, ETC.)

In acute febrile disturbances which are likely to run a short and fierce course, one of the most important considerations from the standpoint of patient, nurse and doctor, is the diet. As a rule the child refuses food and wants drinks only. The tongue is dirty and coated, the breath offensive,

<sup>1</sup> "Post-Vaccinal Encephalitis," Report of the League of Nations Commission, *Lancet* 1925 ii 671.  
<sup>2</sup> Anderson and McKenzie — "Post-Vaccinal Encephalitis," *Lancet* Dec. 3 1912 i 66.



and constipation is present, due partly to confinement in bed but chiefly to dehydration and lack of solid diet

The constituents of the diet which throw least work on digestion, and yet supply it with the calories it demands in the most readily assimilable form, are starch and sugar. In the mouth and intestine starch breaks down into sugar. For these reasons *fever diet* should be along the following lines.

From one to two pints of plain water, sugar water, barley water or well sweetened fruit juice and water, should be taken every 2½ hours (2-4 years of age). It is well to keep a jug of such drinks within the child's sight as an invitation to him to take more. If the child is able to take sufficient quantities at a time the regular times of feeding (breakfast, dinner and tea) should be adhered to, but more frequent feeds may have to be offered. At first, starchy foods (*see p. 402*), made with water, should be administered, since they have a very high caloric value and can be made palatable. They can, of course, be made up with skimmed milk if desired, but certainly not more than half to three quarters of a pint of milk should be given in the day. Bovril or beef tea is useful. Broth made from veal, chicken or beef bones, is recommended, preferably thickened with breadcrumbs or potato or other starchy substance. Plain water ices are indicated.

To sum up, the diet should contain a minimum of fat (butter, cream and eggs) and a maximum of carbohydrate (sugars and starchy foods, such as cereals, bread and potato), with plenty of drinks and a very small quantity of protein. Glucose and barley sugar are both advisable if vomiting is present.

*In chronic febrile conditions*, such as in typhoid, tuberculosis, streptococcal and staphylococcal infections, the patient will, on the other hand, require feeding up almost from the first. The maximum caloric intake which the patient is capable of digesting may be given. (For further details *see each individual disease*.)

## CHAPTER XVII TUBERCULOSIS

TABLE XXV  
MORTALITY DUE TO ALL TYPES OF TUBERCULOSIS  
(England and Wales)

Age	Tuberculosis of the nervous system	Tuberculosis of intestines and peritoneum	Other forms of tuberculosis	Grand totals
Under 1 year	199	1940 38	124	361
1 to 5 years	634	109	260	1 003
5 to 10 years	268	43	128	439
10 to 15 years	189	25	280	494
Totals for 1940	1 290	215	792	2 297
Under 1 year	218	1941 38	133	389
1 to 5 years	880	142	406	1 428
5 to 10 years	378	41	179	597
10 to 15 years	216	33	295	544
Totals for 1941	1 692	254	1 012	2 958
Under 1 year	188	1942 33	118	339
1 to 5 years	685	101	271	1 057
5 to 10 years	287	47	123	457
10 to 15 years	198	57	218	473
Totals for 1942	1 358	238	730	2 326

**Methods of investigating children suspected of having contracted their primary tuberculosis infection**—In every case where a child appears to be wasting, or its health is unsatisfactory for no apparent reason, and especially in cases where there is a history of contact with a tuberculous individual the following investigations should be undertaken by the careful practitioner —

- (a) Tuberculin skin tests
- (b) Search for organisms in the stomach washings or stools
- (c) X rays
- (d) Sedimentation rate

## SKIN TESTS

**Intracutaneous tuberculin test (Mantoux test)**—This is performed by injecting one tenth of one c cm. of a solution of one in ten thousand, one in one thousand or one in one hundred dilution of old tuberculin into the skin just as in the Schick test. A small bleb should be raised. It should be read on the second to fourth day, and the red reaction should be visible up to a fortnight thereafter. It is well to have several dilutions. Where tuberculous infection is strongly suspected the most dilute solutions are first used but if they are negative the stronger solutions should then be tried. This is a slightly painful but extremely accurate method of skin testing. A control solution of glycerin broth should be used at the same time.



Fig. 60.—Positive intracutaneous tuberculin (Mantoux test)

**Tuberculin patch test (Vollmer patch test)**<sup>1</sup>—The test is made by applying a piece of adhesive plaster to the child's cleansed skin. The adhesive plaster consists of two small squares of blotting paper dipped in tuberculin with a similar sized piece of blotting paper between these dipped in glycerin to act as a control (see Plate 21).

The skin of the back must be carefully cleansed with ether or acetone, to remove all fatty substances. The best place to apply the patch is between the shoulder blades to either side of the spine as the child cannot reach this to pull it off. The patch should be left on for 48 hours during which time it should not be wet. At the end of 48 hours it is removed, and the back should be looked at in a further 24 hours and for several days thereafter. A positive test shows very definite erythema in the areas where the tuberculin patches have been present. A negative test should show nothing at

all. A comparison between the Mantoux and the Vollmer test shows the latter to be almost as accurate as the Mantoux. The Vollmer test has the advantage of not being painful and is rapidly and easily applied.

**Tuberculin jelly patch test**—Recently the author has been using a jelly composed of old tuberculin (*Tuberculinum Præparatum*) 2½ per cent inert adhesive 5 per cent. The skin between the shoulder blades is cleaned with acetone and the jelly applied in some characteristic form, such as a 'V' or 'I'. A piece of adhesive (e.g., 2 inches by 1 inch) is then

<sup>1</sup> D. C. Scott. A Comparative Study of the Tuberculin Patch Test and the Intracutaneous Mantoux Test in Childhood, *Brit Med Jov* April 22 1927 1, 874

Fleming (Glasgow), 1943<sup>1</sup> quotes the following figures of the proportion of children at various ages with a positive Mantoux —

0—4 years	9.8 per cent
4—7 "	26.8 "
7—10 "	39.1 " "
10—13 "	44.8 " "

This means that by these various ages the children have had their primary infection. The majority have acquired immunity to a certain degree especially from exogenous infections and their skin has become sensitized by the tubercle bacillus so that they give positive skin tests, apparently for life.

### PRIMARY TUBERCULOUS FOCUS AND PRIMARY COMPLEX

Broadly speaking children may be infected by both the human and bovine tubercle bacillus. The exact proportions are not known but it has been generally accepted that in the vast majority, it is the human bacillus which is responsible.

The primary infection may take place in one of three common sites —

(1) *In the lungs* by far the commonest site, from 90 to 95 per cent of the whole.

(2) *In the bowel wall* giving rise to a tuberculous infection of the mesenteric glands.

(3) *In the pharynx*, particularly the lymphoid tissue of the tonsils and adenoids, giving rise to tuberculous glands of the neck.

### PRIMARY INFECTION OF THE LUNGS<sup>2</sup>

**Mode of infection** —The tubercle bacilli are air borne from infected sputum and are inhaled by the infant or child. The organisms lodge in alveoli, deep in the lung, beneath the pleural surface, and cause the initial lesion, known as the primary focus.

**Source** —Sometimes the source of the infection may be obvious, such as one or other of the parents. Often, the infant is infected by a visiting grandparent who is apparently in good health but has a long standing cough, or by a kindly well meaning neighbour.

**Mode of spread** —The organisms, having gained entrance to the alveoli multiply and irritate the alveolar epithelium producing a localized pneumonic inflammatory process, the *primary focus*. This process may affect a few alveoli only or a whole lobe.

Next, the tubercle bacilli spread from the alveoli along the lymphatics (causing a lymphangitis) to the glands draining the adjacent area. These

<sup>1</sup> G. B. Fleming *Lancet* Nov 6th, 1943, 1, 480.

<sup>2</sup> A. Glenn, *Der Primäre Lungenherd bei der Tuberkulose der Kinder*. Berlin 1912 (English transl. D. Barty King, London, 1916).

Arvid Wallgren, *From Childhood Infection to Adult Type of Pulmonary Tuberculosis, Acta Tuberculosa Scandinavica*, Vol. II, Page 4.

J. W. B. Blacklock, *Tuberculosis in Infancy and Childhood*, with special reference to *Primary Focus*, *Brit. Med. Jour.* Aug. 15, 1936, II, 231.

Arvid Wallgren, *Primary Pulmonary Tuberculosis in Childhood*, *Brit. Jour. Dis. Child.*, May 1933, xlii, 110a.

W. Shelton et al., *Report on the Early Diagnosis of Tuberculosis in Children*, *Arch. Dis. Child.* Sept. 1943, xvi, 137.

are most commonly the broncho pulmonary tracheo bronchial and para tracheal glands. The primary focus, the tuberculous lymphangitis and the glandular lesion, are known as the *primary complex* (Wallgren) (Fig 61)

**Immunity**—During the passage of the organism along the lymphatics and into the glands, the defence mechanism of the body is called into play and lymphocytes wall off the inflamed area. These are the first signs of immunity developing in the body

**Incubation period**—This is the period between the first introduction of the organisms into the body and their passage into the glands with an inflammatory reaction sufficient to cause allergy or the development of hypersensitivity of the skin. This period lasts from three to seven weeks

**Clinical picture during the incubation period**—During these three to seven weeks the child remains perfectly well. There is no cough or wasting or other sign or symptom on which to base a diagnosis. The tuberculin tests are negative and the X ray picture has not sufficiently developed to demonstrate the primary focus. Actually, however, even during this early period, tubercle bacilli may be obtained from stomach washings (Wallgren)

**Allergy**—Coincident with the development of immunity, there is an increased sensitivity to the toxin of the tubercle bacillus. This shows itself by the positive skin tuberculin reactions and such manifestations as erythema nodosum and pleurotenular conjunctivitis

**Anatomical picture of development of allergy**—At the end of the incubation period a sudden inflammatory reaction develops about the primary focus. Hyperæmia, cedema and lymphocytes surround it. The reaction also occurs about the lymphatics and the hilar glands. An X ray will show a definite lesion. The original focus may be lost in the centre of quite a large lobular shadow and other shadows are seen about the inflamed hilar glands (Plat 20). Simultaneously, the skin tests to tuberculin become very strongly positive

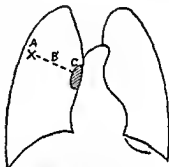


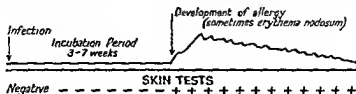
Fig 61  
A Subpleural tuberculous lesion of the alveoli (primary focus)  
B Tuberculous lymphangitis  
C Hilar gland  
A B and C constitute the primary complex

**Clinical picture of development of allergy**—(a) The majority of children, particularly the older ones, remain afebrile and show no clinical upset. The lesion may be so slight that they do not even develop a cough and it heals completely and gives rise to no symptoms throughout life

(b) A smaller proportion run a benign fever lasting from one to four weeks. The symptoms are those of a cold or mild influenza and the temperature may rise at the beginning to  $101^{\circ}\text{F}$ . The majority of cases subside in a fortnight. In addition to fever, other symptoms which develop are lassitude, headache, loss of appetite and weight, and a cough. The cough may be a peculiar stridor, especially in infants, or a hoarse cough suggesting mediastinal glands. The physical signs on examination are

extremely meagre, and this cannot be too often stressed. An ailing child with unilateral bronchitis should always be suspected of tuberculosis. The lesion may be in the central portion of the lung, with healthy lung tissue over it, and neither by percussion nor by auscultation can gross abnormality be detected. The positive tuberculin test, however, should lead to radiography of the chest, with correct diagnosis.

(c) In a small proportion of cases as the fever and skin sensitivity or allergy develop so erythema nodosum appears. This syndrome is a great help in the diagnosis of the primary infection.



**Course and treatment of the primary tuberculous infection.**—In the slight cases in older children the whole process passes unnoticed. Such individuals seem to have gained immunity for life from this slight infection. In the more severe cases and in the very young the fever subsides in 2 to 4 weeks, but such a child should be kept in bed until the temperature is normal. Fresh air, good food, and tonics are essential. It may be several weeks before the sedimentation rate has returned to normal. A good working rule is to keep such a child under observation for a period of three to six months lest there be a recurrence or fresh exacerbation of the infection.

The advisability of this is shown in the following table giving the interval between the primary infection and the occurrence of tuberculous meningitis —

Interval	Months								Total
	0-1	1-2	2-3	3-4	4-5	5-6	6-7	7-13	
Cases	15	20	12	4	2	3	2	1	64

From this it appears that most cases of tuberculous meningitis occur 1 to 2 months after the first manifestations of primary tuberculosis. It seldom occurs later than 3 months after the primary infection has shown itself. These 3 months should be looked upon as the danger period.<sup>2</sup>

**What happens to the primary focus?**—(1) In the vast majority of cases the lesions in the lung, parenchyma, lymphatics and hilar glands calcify and are surrounded with fibrous tissue, and almost disappear.

(2) In a small proportion of children, however, an acute infection such as measles may reactivate the primary complex and a sudden extension of the disease results. This very often occurs at puberty and under the age of 2 which are danger periods when the child is especially susceptible to such an extension.

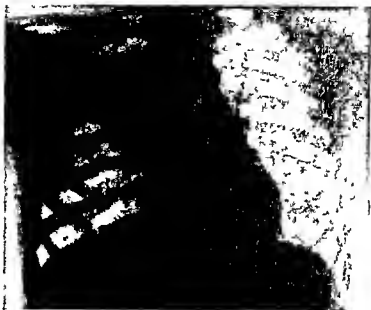


Fig 1.—Primary pulmonary tuberculous infection. Male aged 6 years 10 months. Showed listlessness and loss of weight though not particularly ill. Positive Mantoux. Sister, aged 2 years, died of tuberculous meningitis two months later.



Fig 2.—Miliary tuberculosis in a child, aged 6½ months.

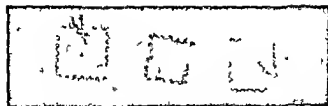


Fig 1 Three squares of filter paper on adhesive plaster: the outer squares are saturated with undiluted old tuberculin, the centre square (control) with glycerin broth.



Fig 2—A positive reaction twenty four hours after removal of the patch

Plate 21 Tuberculin "patch test."  
*(By courtesy of Dr Donald Court)*



(3) Lung collapse and bronchiectasis from pressure on the bronchi, due to enlarged hilar glands, may occur in some cases

(4) There may be an extension of the infection within a few weeks or months of the establishment of the primary lesion. In some cases, the extension may occur many years later

The disease may progress by —

- (a) Direct extension of the lesion to the surrounding parts or
- (b) Hæmatogenous spread (Miliary tuberculosis)

#### (A) DIRECT EXTENSION OF THE LESION

This may give rise to

(1) Pleurisy, by a spread from the primary focus, from a tuberculous broncho pneumonia, or the emptying of a caseous hilar gland into the pleural cavity

(2) Active tuberculous lobar pneumonia with caseation and cavitation as seen in adults

(3) Eпитuberculosis, where a caseous gland with low virulence bacilli is emptied into the bronchus and scattered throughout one lobe of the lung giving rise to a consolidation of that lobe

#### TUBERCULOUS PLEURISY

**Ætiology and pathology.**—Probably an inflamed or caseous subpleural primary focus or a caseous hilar gland erupts into the pleural cavity and thus infects it. Clear straw coloured fluid collects. The fluid contains lymphocytes, and later it may be milky or opaque. Both the visceral and parietal pleurae are thickened, red, and baggy, with a tendency to form adhesions between the adjacent surfaces

**Symptoms and clinical picture**—The patient usually complains of a sharp pain in the affected side, and there is fever and a persistent cough. Friction may be heard at the commencement, but usually the absent breath sounds and dullness to percussion, with displacement of the heart, suggest the presence of fluid. The blood count is usually normal, as opposed to the well marked polymorphonuclear leucocytosis found in purulent empyema. The skin tests give a positive reaction to tuberculin, and an X ray of the chest bears out the clinical diagnosis

**Prognosis and treatment**—The prognosis is usually excellent. The chest may be strapped with broad bands of adhesive during the painful stage, but they should be removed when that stage is passed. The child should be kept in bed and nursed in the fresh air if possible. If the effusion is gross, and there is much displacement of the heart so as to occasion distress, it may be necessary to withdraw fluid with a needle and syringe. Usually this is not necessary, however, and the fluid is gradually absorbed.

As the fluid disappears the source of the pleurisy is usually visible in the X ray. Further X rays should be taken together with sedimentation rates to ensure that the lesion is completely healed. Usually a few adhesions remain but these may be so placed that very little permanent functional disability results.

#### ACUTE TUBERCULOUS PNEUMONIA

Occasionally, after the primary infection the primary lung lesion and adjacent infected glands show no sign of healing. The primary site may

spread and involve the whole lobe of the lung or the hilar glands caseate and discharge virulent bacilli into the adjacent bronchus

**Clinical picture**—This adult type of the disease is comparatively rare in early childhood, being more often found at or after puberty. The fever is high and swinging and there is persistent cough with sputum especially when cavitation has occurred. The physical signs are those of consolidation as seen in the adult. This is the true case of 'consumption' known to the public.

**Prognosis and treatment.**—The outlook in these rapid and virulent cases is extremely bad and death from hæmoptysis, exhaustion or generalized infection may terminate the case. Artificial pneumothorax should be tried. Rest in bed, fresh air and good food and tonics are indicated. Because of the possibility of a mixed pneumococcal or streptococcal infection a course of one of the Sulpha drugs is indicated and may be helpful.

#### EPITUBERCULOSIS<sup>1</sup>

There has been much speculation on the pathology of this clinical picture. Since these children almost invariably recover autopsies have been very few indeed. It is generally accepted that a caseous gland containing low grade avirulent tubercle bacilli erodes a bronchus and discharges its contents into the lumen. They are coughed out towards the periphery of the lung and set up a very mild infection and reaction which spreads gradually to the periphery. Occasionally this picture may be simulated by a collapsed lobe of the lung due to an enlarged gland pressing on the bronchus. The upper lobes are much more commonly affected than the lower lobes in epithuberculosis.

**Clinical picture**—It is typical of the condition that it is unsuspected and discovered by accident. The child has no temperature and appears to be in reasonably good health. The physical signs are those of lobar consolidation—dullness on percussion and tubular breathing on auscultation. The physical signs and radiographic picture of lobar pneumonia last for many weeks or months and then appear to clear up completely or leave minor evidence of fibrosis or calcification only. In such cases the tuberculin skin tests are strongly positive. The treatment is similar to other tuberculous infections—rest in bed with good food, fresh air, and tonics such as cod liver oil and malt.

#### (B) HÆMATOGENOUS SPREAD OF TUBERCULOUS INFECTION

It is claimed that there is some spread to the blood stream via the thoracic duct in all cases of primary infection. Gross hæmatogenous infection, however, occurs when a caseous gland has eroded the wall of a vein and emptied its contents into the vein either all at once or at intervals. The organisms pass to all parts of the body, but certain situations are more commonly affected than others. These are—

- (a) The lungs, where miliary tuberculosis occurs
- (b) The brain and meninges, giving rise to tuberculous meningitis
- (c) The bones and joints, giving rise to latent or active infection

<sup>1</sup> Eliasberg & Neuland *Jahrb. f. Kinderk.* Berlin 19 0 xliii 82 and 1901 xiv 173  
J. C. Spence *Arch. f. Child.* 1917 viii 1

L. G. Garmon, The Childhood Type of Tuberculosis, *Lancet* May 26 1931 i, 1101

## MILIARY TUBERCULOSIS

**Ætiology and pathology.**—Miliary tuberculosis as a primary lesion does not exist for practical purposes. In every case a primary focus will be found somewhere. In a series of 70 post mortem examinations of cases of tuberculous meningitis (see p. 228), 87 per cent showed a primary lesion in the gland at the bifurcation of the bronchi and 19 per cent in an infected alveolar gland. These infected and usually caseous glands tend to burst into some vessel near by, disseminating tubercle bacilli throughout the body via the blood stream. The commonest site is the meninges giving rise to tuberculous meningitis, but concurrent with this and almost as common, is involvement of the lungs. In 75 per cent of cases the spleen, kidneys and liver were infected, and in 45 per cent there were ulcers in the bowel.

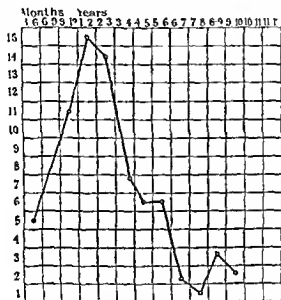


Fig. 62.—Age incidence of miliary tuberculosis (tuberculous meningitis) in 70 post mortem examinations (East & Hart).

**Age-incidence**—The disease is most commonly found between the ages of one and three years (see Fig. 62).

**Seasonal incidence**—The largest proportion of the cases is in the first quarter of the year, the explanation being that at this time infectious diseases, such as measles, whooping-cough and scarlet fever are rife. Moreover, in winter the weather is bad, confinement indoors is at its maximum, sunshine is scarce, fresh vegetables are difficult to obtain and cows being stall fed at this time, give milk deficient in vitamins.

**Clinical picture**—If the brunt of the infection has fallen on the lungs the symptoms are largely respiratory. The respirations are quickened, the child becomes cyanosed and restless, the pulse is extremely rapid, there is wasting, slight cough, lack of appetite and extreme irritability. If

the brunt of the infection falls rather on the meninges, the symptoms are general lassitude, head ache, drowsiness and irritability (see p. 228).

On examination probably the most significant feature of the case is the paucity of physical signs, although the child is obviously ill. There may be a few moist sounds scattered over the chest, but nothing definite. On palpation of the abdomen, especially when the chest is mainly involved the spleen can often easily be felt, a very significant finding. The temperature may be continually up, but in the early stages there are short bouts of pyrexia only. The rapidity of the pulse and respiration, with a tendency to cyanosis, is, however, a constant feature which should not be overlooked.

**Diagnosis.**—This is extremely difficult, especially early in the disease, because of the lack of physical signs. An X ray of the chest shows typical fine, mottled or stippled appearances. On washing out the stomach or examining the stools tubercle bacilli may be discovered. With involvement of the meninges, meningitis is



Fig. 63.—Miliary tuberculosis in a child aged 6½ months.

quickly suspected and proved.<sup>1</sup> Very often typhoid or paratyphoid is suspected, but can be ruled out by a negative Widal test. Examination of the urine excludes pychitis, the general appearances of which may somewhat resemble miliary tuberculosis. In the early stages the Mantoux or tuberculin patch tests are positive. In the late stages, however, these tests may be negative, and the sedimentation rate may even be normal. (Plate 21.)

**Prognosis and treatment.**—Miliary tuberculosis is usually fatal. The treatment is therefore palliative. Oxygen relieves the dyspnoea and cyanosis, and for meningitis lumbar puncture should be performed.

**Prevention.**—Since miliary tuberculosis is always secondary to a primary focus, there is no reason, once the primary focus has appeared, why the disease should not be arrested and its spread throughout the body prevented. With the development of the primary lesion the child shows such symptoms as lassitude, wasting, lack of appetite, dry skin and hypotonia of the muscles. An investigation with X-rays and the intracutaneous tuberculin test (p. 332) may reveal the site of the primary focus at this stage. The general health should be raised, an abundance of fresh air, a change to the seaside, good food and sunlight are essential. (See p. 344.)

After the age of six, miliary tuberculosis is comparatively rare, and therefore the greatest care should be maintained until this age has been reached.

**Tuberculous meningitis.** (see p. 228)

attain or even exceed the size of an orange; they caseate, liquefy and finally break down and discharge, as a rule, at the umbilicus.

**Tuberculous peritonitis. Ascitic form.**—Tubercles may appear on the peritoneal surface, having spread from ulcers in the bowel or from a caseous gland which has discharged into the abdominal cavity, and these peritoneal tubercles spread rapidly from the bowel surface to the parietal peritoneum. A clear effusion forms in the abdominal cavity and the loops of bowel float about in this fluid. There may be very few adhesions between the bowel loops at this stage. It is when healing commences, or after an operation when the effusion has been released, that the loops of bowel tend to adhere both to one another and to the parietal peritoneum. This is the so-called *plastic form of peritonitis*. The omentum may become grossly involved, the tubercles coalescing and actual caseous masses forming in it. The omentum tends to be drawn upward over the transverse colon, and a knobbly, doughy mass can be felt in the abdomen. In the plastic type there is a great tendency for loops of bowel to become adherent to the abdominal wall and for the bowel contents to burrow through and burst, drawing at the navel—so called *fecal fistula*. Obstruction often occurs in this type so that vomiting and abdominal pain are common. Perforation of an ulcer resulting in septic peritonitis, is commonly the cause of death.

**Symptoms and physical findings.**—Probably, the commonest complaint of a child with a tuberculous infection anywhere in the abdomen is pain. In *tuberculous ulceration of the bowel* the child may remain apparently perfectly fit physically, but in prolonged, severe cases rapid wasting occurs. The stools are loose and contain much mucus. They are most offensive and often light coloured, as they are for instance, in *cœliac disease*, in which there is some clinical resemblance. In slight cases the temperature is not raised but in severe cases there is a swinging temperature varying between 101° and 99°.

In *tuberculous mesenteric colicky pains* in the abdomen, lassitude and failure to gain weight are the common symptoms. On palpation little may be made out unless the child be asleep, or under an anæsthetic, or will allow the abdominal wall to relax completely. When this is done the glands may be found. Sometimes there is a group situated at the root of the mesentery, in which case they are fixed, at other times the glands are freely movable lying in the mesentery itself. Unless the lesion is extensive there is no fever.

In the *ascitic form of tuberculous peritonitis* colicky pains may be present directing attention to the abdomen, which is noted to be large and to contain fluid. It may be greatly distended and shiny, the vessels prominent over the surface. An examination may reveal dullness of the flanks, due to the fluid, and a mass of enlarged glands and rolled up omentum may be felt on deep palpation. The child itself is wasted, anæmic and languid. There is no fever unless the infection is generalized.

In the *plastic form of peritonitis* the abdomen feels doughy and the loops of bowel are bound one to the other. The whole abdomen is too firm and with relaxation of the abdominal wall, large caseous glands and the rolled up omentum are easily made out. Very often there is an irregular temperature, especially when the patient is not progressing well. There may

stomach and bowel. Finally, the control of the child and its habits is rendered easier, with the result that nursing becomes much simpler.

Apart from the damage to the body itself from absorbed toxins in *tuberculous ulceration of the bowel* there is a much greater damage by malabsorption, due to hurry of the food through the bowel and imperfect digestion. This chronic intestinal indigestion must be treated by offering the bowel those foodstuffs which are most easily digested and assimilated. A diet high in protein, low in fat and moderately high in carbohydrates is therefore the most suitable and since in *tabes mesenterica* and *tuberculosis peritonidis* there is very often an accompanying ulceration of the bowel such a diet should be adopted in these conditions also. Not only are the proteins the best managed element of food but they produce alkaline, constipated stools, which is an advantage. The diet should contain raw, scraped or underdone steak, a little well skimmed boiled milk, or junket made from skimmed milk, two or three eggs in the week, minus most of the yolk, and crisply done bacon from which most of the fat has been removed. The fat being difficult to digest, is kept low. Hence the skimmed milk and the egg without yolk. Carbohydrates are best given as starches, and then in a most digestible form. Bread should be given as crisp toast or rusks, or it should be re-baked in the oven. Potato should be limited to one small one in the day. Milk pudding should be well cooked. This diet is very much like that given for cœliac disease (see p. 86).

As the child improves and the stools become normal, a more liberal supply of fats (butter, milk cream and eggs) should be given.

**Sunshine**—Real or artificial sunlight is a most valuable therapeutic agent in tuberculous infections, especially abdominal tuberculosis. It must be administered in carefully graduated doses starting with extremely little. The aim should be to get the abdomen and in fact the whole child, sunburnt. In patients who exhibit pigmentation, the prognosis is much better than in those who do not. Exposure to fresh air is also an advantage and on this account sea air is better than inland air other things being equal. When the weather permits, the child's clothes should be reduced to a minimum and the limbs exposed to the direct sunlight and fresh air during part of the day.

Probably no therapeutic measure is more important in this disease than the administration of cod liver or halibut liver oil. It appears to raise the immunity to the tubercle bacillus and to induce healing particularly when the oil is well borne. Since a high fat diet is not well borne, the vitamins must be administered in as concentrated a form as possible. Codeine,  $\frac{1}{20}$ th of a grain three times daily, is useful in older children.

**Prognosis**.—Abdominal tuberculosis is gradually becoming less common. There is a saying that the children under two die and those over two live (for statistics, see p. 831). Broadly speaking, this is undoubtedly true. Certainly, the younger the child the more the infection tends to become generalized. The mortality becomes comparatively small over the age of three. In *tabes mesenterica* where the glands caseate, liquefy and drain at the umbilicus, or where a faecal fistula has developed, the outlook though grave, is not hopeless, and a large proportion of the cases recover. If the ulceration of the bowel is extreme, there is a tendency to stenosis

afterwards, with chronic ill health and intermittent obstruction. In *tuberculous peritonitis*, unless health is extremely low and the infection becomes generalized the outlook is excellent. The ascitic form answers readily to simple drainage, and the plastic form clears up entirely with the general treatment outlined.

### 3 TUBERCULOUS CERVICAL ADENITIS

In *tuberculous cervical adenitis* the primary lesion is as a rule situated in the lymphoid tissue of the tonsils and adenoids.

**Ætiology and pathology.**—The cervical glands most commonly infected are those which lie at the angle of the jaw and drain the tonsil. As a rule, there have been one or two attacks of sore throat, with the result that the tonsillar glands and some of the other cervical glands have become permanently enlarged. There may be an interval of some weeks or months when there is a constant increase in the size of the glands on one or both sides. This is due to an infection with the tubercle bacillus which has gained entrance through the damaged tonsil or adenoid tissue and has reached the already affected gland. Inflammation occurs and may rapidly pass on to a cold abscess, or healing may be followed by caseation. The infection may not always manifest in the tonsillar gland. In some cases the glands deep or posterior to the sterno-mastoid may be the first to be involved, and usually several become enlarged.

**Clinical picture.**—The child with slightly enlarged cervical glands may be discovered to have a large swelling half the size of a tangerine orange, over the side of the neck. This swelling may appear slowly, or in the course of a few hours and there may or may not be a temperature, usually there is not. On slight pressure over the swelling no great tenderness is complained of and at first, if the swelling is deep seated there is no fluctuation. Later, as the pus nears the surface, the skin becomes red and shiny and distinct fluctuation is noticed. If no treatment is instituted the skin breaks down and a sinus is formed. Differentiation between acute streptococcal or pneumococcal cervical glands and tuberculous glands may, clinically, be very difficult. In these circumstances the Mantoux or the patch test (p. 332) may give valuable assistance.

**Prognosis.**—The author may have seen extremely unfortunate, but it has been his experience that nearly all cases of proved tuberculous cervical adenitis come to surgical removal or breakdown.

**Treatment.**—Cervical adenitis should always suggest the possibility of tuberculosis.

**Removal of septic foci.**—The first step is the removal of septic teeth and tonsils. It is usually through the tonsils that the glands become infected.

**Conservative treatment.**—Splinting the neck, as in acute cervical adenitis (figs. 19, 20 p. 137), is indicated and it should be done in the same way. When head movement is prevented, the cervical glands are not pressed upon by the neck muscles and thus obtain complete rest. Should the glands not react favourably to conservative treatment, and if

<sup>1</sup> Sir L. Barrington Ward, "Tuberculous Glands of the Neck in Children," *Lancet* April 21 1931, 1, 280

there is a tendency to matting of the glands and if cross formation surgical aid should be called in before the process has gone too far. The work of the surgeon is made much more difficult if he must deal with the situation when inflammation is well advanced.

**Sunshine and ultra violet rays**—Exposure of the affected area of the body to sunshine or ultra violet rays is strongly recommended if carried out with care. As in pulmonary tuberculosis this powerful therapeutic agent must be used with caution.

**General treatment**—Attempts should be made to improve the patient's general health by a change to the seaside, a well balanced diet, a maximum of fresh air and rest with some suitable cod liver or halibut liver oil preparation which does not upset the digestion.

Dr Gordon Pugh<sup>1</sup> and others have praised the excellent results of applying radium to tuberculous glands in the neck. A high proportion of apparent cures or improvements has followed.

**Prophylaxis against tuberculosis with B.C.G. Vaccine**—Wallgren<sup>2</sup> working in Gothenburg claimed that B.C.G. vaccine was both safe and efficient. The vaccine was made from living bovine tubercle bacilli which by the method of preparation were rendered avirulent.

He removed the infants or children of actively tuberculous parents and kept them under observation. If they continued to give a negative tuberculin test for six weeks then they were considered as not yet having been infected. New born infants removed at once from their mother were also considered as not infected.

The infant or child was then given 0.05 mgm. of the vaccine intracutaneously and a small abscess the size of a pea formed and later discharged. After roughly six weeks the tuberculin skin test yielded a positive result, and the patient returned to the tuberculous parent it being assumed that the child had now acquired a considerable degree of immunity.

Wallgren had 355 children under observation for varying periods. No child died of tuberculosis in this series. He did not claim that this vaccination fully protected the child but considered that it did so to about the same extent that a primary infection with tuberculosis would.

As a result of the vaccination of children of parents with open pulmonary tuberculosis the death rate from tuberculosis in Gothenburg was reduced to about one-thirteenth its previous figure in a period of five years. This method of protecting children might well be applied in this country.

<sup>1</sup> *Proc Roy Soc Med (Sec Surg)* N. II Nov 3 1922, xxii 10.

<sup>2</sup> A. Wallgren "Value of Calmette Vaccination in Prevention of Tuberculosis in Childhood" *Journal Amer Med Assoc*, Nov 3rd 1921, clii 3541.



## CHAPTER XVIII

### SYPHILIS IN CHILDHOOD

In the vast majority of cases of syphilis in childhood infection takes place before birth. These are termed congenital syphilis. In rare cases however, syphilis is acquired after birth (*extra genital infections*).

TABLE XXVI

*Cases of Congenital Syphilis dealt with for the first time at the Treatment Centres*

Year	Under 1 year	1 and under 5 years	5 and under 15 years	15 years and over	Totals
1931	339	291	674	922	2 430
1932	302	180	857	805	2 144
1933	300	157	774	760	2 016
1934	298	165	708	839	2 008
1935	251	160	671	944	2 031
1936	241	132	600	935	1 908
1937	211	144	534	940	1 829
1938	216	123	448	901	1 738

TABLE XXVII

*Death rates per 1 000 live births of infants under one year certified as due to syphilis —  
Extracted from the Registrar General's Statistical Reviews (England and Wales)*

1912	1 34	1927	0 77
1913	1 46	1928	0 71
1914	1 53	1929	0 64
1915	1 44	1930	0 50
1916	1 57	1931	0 51
1917	2 07	1932	0 47
1918	1 90	1933	0 40
1919	1 76	1934	0 34
1920	1 51	1935	0 29
1921	1 43	1936	0 27
1922	1 12	1937	0 23
1923	1 01	1938	0 20
1924	0 91	1940	0 147
1925	0 82	1941	0 199
1926	0 84	1942	0 110

### CONGENITAL SYPHILIS

**Ætiology**—Syphilis is the result of an infection by the *Spirochæta pallida*. In most cases the father has contracted syphilis and has passed on the infection to the mother who has given birth to a syphilitic infant. It is an interesting fact that very often the mother shows no clinical signs of the disease although she may have a positive Wassermann reaction. It is argued by some that this is because she has harboured a syphilitic placenta and has thus developed immune bodies. The mother may become infected by a primary vaginal infection or through an infected placenta. It is assumed that the ovum is fertilized by an infected spermatozoon. The Wassermann reaction may be applied to both mother and child but as a rule if it is positive in one it is positive in the other also. Although the Wassermann reaction in the father may be negative this does not prove that he was not the primary infecting agent, as he may have had treatment after infecting his wife.

Syphilitic infants are not highly infectious to other children. There is no doubt that infection can be carried, but instances appear to be extremely rare. The suckling of a syphilitic infant by a wet nurse is likely to produce breast infection, but the mere handling of the child is not likely to lead to infection.

**Clinical picture.**—A syphilitic infant may be born dead either at full term or prematurely. It may be a *macerated fetus*, the placenta being infected, friable and containing infarcts. A history marked by such events as the following is quite common: first an abortion at 2 or 3 months, then possibly another at 5 months, then a premature birth or stillbirth at 7 months, and finally a full term child apparently quite healthy. At birth, as a rule, the child shows no signs of syphilis. In a very small percentage only the rash is present at birth, or develops within the first week; in the great majority it develops between the fourth and eighth weeks. The rash may be a *macular* one, which appears on the buttocks, the palms of the hands, the soles of the feet and the legs; it is pale-red or copper coloured, and has a shiny, dry, silky appearance. It may be present over the whole body, but this is uncommon.

The syphilitic *pemphigus* which occurs on the soles of the feet, palms of the hands, the knees and sometimes the face, does not usually appear for two or three weeks, but occasionally the infant is born with it. There may be a *syphilitic wig*, that is, the hair



Fig. 64.—Congenital syphilis showing condylomata about the anus  
(1/2 inch of 1/2 x 1/2 inch).

may be thick, black and straight, but this is of no great importance, as many normal infants have a profuse growth of hair. *Snuffles* is one of the earliest symptoms; it may be present at birth or appear within a few days, or develop after some weeks. Snuffles is due to a syphilis of the septum and turbinates, in which other organisms take part, causing a muco-purulent discharge and destruction of the nasal bones. The bridge of the nose then falls in, and the flat saddle shape nose results. *Rhagades*, or scarring about the mouth, in its most typical form is best seen in older children. In the younger child it assumes the picture of a scra about the mouth or merely roughness. *Cenilyomatous* form at the angles of the mouth and anus (Fig. 64). In older children a syphilitic perforation of the soft palate (Fig. 65) may occasionally be seen. In little infants with syphilis the cry is often hoarse, due to syphilitic laryngitis. In about half the cases there is an enlargement of the spleen within a few weeks of birth. A new-born infant with an enlarged spleen, whether it has any of the other manifestations of syphilis or not, is suspect; combined with other manifestations of syphilis this renders the diagnosis almost certain.

**Syphilis of the bones.**—One of the earliest manifestations of syphilis is that of Pott's pseudo-paralysis. The child develops acute syphilitic epiphyseitis, usually at the upper end of the humerus, but it may be elsewhere. The arm is not used, and the question is raised whether a fracture may have occurred at birth unnoticed. As a rule, however, there are other manifestations of syphilis which help in the diagnosis.

The commonest lesion in congenital syphilis is osteitis, best seen in the tibia, which show bowing and curving forward and in thickening of the skull bones especially bossing over the frontal and occipital regions. A syphilitic dactylitis shows a typical periostitis in the skelogram. (See Plate 22)

**Syphilitic meningitis**—Cases of syphilitic meningitis as a rule manifest themselves by hydrocephalus which is usually external. The head becomes larger and larger the spleen may be palpable and the child may show other signs of syphilis. The Wassermann reaction will show blood or cerebro spinal fluid to be positive. It is always well to test the mother at the same time. The post mortem investigation of syphilitic meningitis shows much thickening of the meninges with a tendency to a thienons and cystic spaces into which hemorrhages may take place. The choroid plexus, functioning normally has produced the cerebro spinal fluid but the inflammation of the meninges has prevented the fluid from draining away resulting in hydrocephalus. The cerebro spinal fluid may be bloodstained but as a rule it is moderately clear. The albumin is raised sugar is normal and lymphocytes are increased (see p 220)



Fig. 65—Syphilitic perforation of the palate

**Lesions of the eyes**—The commonest lesion is choroiditis, when black or dark patches may be seen scattered over the choroid. This is present at any age. Interstitial keratitis comes on as a rule during second dentition between the ages of six and twelve. With photophobia, opacities of the cornea and peg shaped teeth or some other manifestation of syphilis the diagnosis is comparatively easy.

**Mental deficiency**—A certain small proportion of congenital mental defects are due to syphilitic infection. The vast majority however are hydrocephalic.

**Syphilitic testis is rare**. A Wassermann test should always be done in every gross enlargement of the testis in childhood.

**A hard, enlarged liver may be felt**. There may be two processes at work (a)iliary gummata with fine intercellular cirrhosis, (b) localized patches of gummatous material often just beneath the capsule. It is said that the left lobe of the liver is more often involved in syphilis of childhood than the right. Extreme jaundice due to syphilis is a great rarity in childhood.

In rare cases syphilitic carditis is found

White pneumonia in the new born infant is also extremely rare—there is an interstitial fibrinosis of syphilitic origin involving the whole of one lung.

Syphilitic nephritis comes in as a rule between the fourth and eighth months and often proves fatal. Edema is marked especially in the face (Fig. 66); the urine is scanty and contains much blood, albumin and many casts. Very often some other



Fig. 66.—Case of syphilitic nephritis aged 6 months showing generalized edema.

syphilitic manifestation is present. *Parotitis* the kidneys are found filled with milium gummata. *Parotismal horn glaucoma* is nearly always a syphilitic manifestation. Chills will bring on an exacerbation and the urine is bright red because the cells have all been laked, colouring the urine with haemoglobin.



Fig. 67. Congenital syphilis showing Hutchinsonian teeth (1 year and 6 months of the subject).

The teeth.—Peg shaped or Hutchinsonian teeth (Fig. 67) are extremely characteristic of congenital syphilis. These appear in the second dentition only. The broadest portion of the peg is next the gum, the free edge being narrow in all dimensions, with a characteristic crescentic notch. Carious teeth however often wear to such a shape and may be mistaken for syphilitic teeth.

**Summary.**—Congenital syphilis manifests itself in infancy in a rash, snuffles, pseudo-paralysis, occasional nephritis, condylomata about the mouth and anus, and hydrocephalus. The later manifestations of syphilis are interstitial keratitis, saddle shaped nose, Hutchinsonian teeth, painless bilateral swellings of the knees (Fig. 68), perforation of the palate, cirrhosis of the liver, sabre shaped tibiae, osteitis and periostitis of the various bones, and bossing of the skull.



Fig. 68.—Case of congenital syphilis, aged 8 showing bilateral enlargement of the knees bossed skull and depressed bridge to nose.

**Pathology.**—The *Spirochæta pallida* causes an inflammation near the blood vessel, the reaction to which shows the presence of round cells, and, later, fibrosis occurs. The spirochæta is best demonstrated in the liver, but a confirmation of the diagnosis depends largely on the Wassermann reaction.

**Treatment.**<sup>1</sup>—Just as in adult syphilis, the longer the lesion has existed the harder it is to eradicate the infection from the body, and the more likelihood there is of permanent damage. In congenital syphilis the infection was acquired *in utero* and had existed for some months before the child was born. The infection is therefore very widely spread throughout the tissues and some of the vital organs are already grossly damaged.

Of the preparations available the three most used in children are neoarsphenamine, sulpharsphenamine and bismuth oxychloride. The dosage is as follows:—

#### INITIAL AND FINAL DOSES

Age	Neo Arspen- amine (Intravenous)	Sulpharsphen- amine (Intramuscular)	Bismuth oxy chloride (Intramuscular)
1 mo. 1 yr.	—	0.02-0.15 gm.	0.1 c.cm. 1 c.cm.
1-3 years	0.1-0.3 gm.	0.05-0.3 gm.	0.25 c.cm. 1.5 c.cm.
3-12 years	0.15-0.45 gm.	0.1-0.45 gm.	0.5 c.cm. 2 or 2.5 c.cm.

Six injections are given at intervals, gradually increasing from the initial to the full dose.

**Technique of administration.**—Sterilized distilled water, 1 to 3 c.cm., is used to dissolve the preparation. Neoarsphenamine is best given intravenously. Sulpharsphenamine may be given subcutaneously or intramuscularly. Injections directly into the muscle of the buttock are almost painless. When the preparations are given intravenously a vein at the bend of the elbow or at the ankle should be chosen. There is no better syringe than the all glass three piece syringe sold by Burtoughs, Wellcome and Co. The longitudinal sinus should not be used for arsenic preparations, but 1 blood for the Wassermann test may be taken from it.

**Treatment by mercury.**—Hydrargyrets may be given by the mouth in doses of  $\frac{1}{2}$  grain to 2 or 3 grains twice daily. In addition mercuric ointment may be used a small piece the size of a pea being rubbed into a different portion of the child's body each day. These forms of treatment should be continued until the clinical manifestations have cleared up until there is no further recurrence and until the blood Wassermann test is negative. Mercury should not be depended upon alone, but should be an adjunct of one of the arsenic preparations.

Children in whom the Wassermann reaction gives a positive result but who exhibit no symptoms or signs of the disease should be treated prophylactically. A syphilitic mother who is pregnant should be treated during pregnancy in order that her infant may be born free from syphilis.

It is claimed that penicillin is efficient in the treatment of this disease (see p. 385).<sup>2</sup>

**Prognosis.**<sup>3</sup>—Infants in whom there are gross manifestations of syphilis at or shortly after birth show an extremely high mortality and even if they survive, exhibit, as a rule, some permanent disability. If the manifestations are delayed for some weeks or months and treatment is begun immediately they do appear, there is good hope of the child being quite cured, that is the clinical manifestations clear up, there is a permanently negative result to the Wassermann reaction, and no further recurrences take place. Of all the manifestations, eye symptoms are the most difficult to eradicate. In the mentally defective child improvement but not recovery takes place. It is obvious that the ultimate prognosis depends entirely on the organs affected and the duration of infection before treatment is instituted.

<sup>1</sup> D. N. Naborn. The Treatment of Congenital Syphilis in Children, *Lancet*, June 6, 1940, i. 1138.

<sup>2</sup> *Brit. Med. J.* or Mar. 23 April 15 1941, 4, 478-523.

<sup>3</sup> Donald Paterson. Transmitted Congenital Syphilis, *Brit. Jour. Child Dis.*, Oct.-Dec., 1939, xvii. 127.

## CHAPTER XIX

### RHEUMATISM<sup>1</sup>

THE rheumatic state includes a variety of manifestations of rheumatism affecting various organs and differing widely in the clinical picture to which it gives rise. Since we have no certain test or criterion the diagnosis is extremely difficult and therefore is very often wrongly made. With the exception of heart disease, nodules and chorea, few of the manifestations can be taken as definitely rheumatic.

TABLE XXVIII<sup>2</sup>

INCIDENCE.—England and Wales. Deaths from rheumatic fever and heart disease at ages under 15 years before the war and since the outbreak of war.—

Year	Totals Under 15 years				0-5 years				5-15 years			
	Rheumatic Fever		Heart Disease		Rheumatic Fever		Heart Disease		Rheumatic Fever		Heart Disease	
1932	377		690		35		60		342		630	
1933	493		771		58		49		435		722	
1934	570		893		51		62		470		831	
	M	F	M	F	M	F	M	F	M	F	M	F
1939	129	172	512	474	15	14	54	34	114	158	458	440
1940	120	143	380	481	10	16	33	46	110	127	342	435
1941	95	94	344	424	16	8	48	36	79	86	296	398

**Ætiology. Organisms.**—For 25 years a controversy has raged on the ætiology of rheumatism. Evidence attributing it to an infection was first put forward by Poynton and Paine<sup>3</sup> who claimed to have made the discovery of a diplococcus in the heart's blood, in nodules, in damaged valves and, later, in cases of chorea, in the brain. Various workers have entirely failed to substantiate this, while some have, in part, borne it out. There can be little doubt that, whatever the organism be, this disease is due to an infection, and everything suggests that the infection gains entrance through the tonsils. Once the infection has taken place it cannot be said to have been eliminated, although many years may have elapsed since clinical onset.

<sup>1</sup> Report of Public Health and Medical Statistics No. 41. Acute Rheumatism in Children in relation to Heart Disease. (H.M. Stationery Office 1934).

<sup>2</sup> The Health of the Schoolchild. Annual Report of the Chief Medical Officer. 1941 and of Public Health for the year 1934 (H.M. Stationery Office).

<sup>3</sup> J. Poynton and L. Paine, *Lancet*, 1909 II 801-802.

Workers<sup>1</sup> at Great Ormond Street have demonstrated an ultra microscopie organism in the pericardial fluid and pleural exudate from cases of rheumatism. These suspensions are agglutinated by the sera of patients. They were also able to demonstrate streptococcal precipitins in the blood of rheumatic patients following acute streptococcal throat infections, the most usual time for them to appear being about the tenth or thirteenth day, the so-called "silent period."

**Sex-incidence**—Statistics show the disease to be rather more common in females than in males but with a great difference in the manifestations in the two sexes. Chorea is more common in girls than in boys whereas arthritis is more common in boys than in girls. Heart disease is equally common in the two sexes.

**Heredity**—A history of an hereditary tendency can be elicited in about half the cases.

Wilson and others<sup>2</sup> 1943 studied 688 rheumatic children and came to the conclusion that the most important factor in the pathogenesis of rheumatic fever was the genetic susceptibility of the host. They came to the conclusion that heredity is primarily responsible for the familial incidence of the disease, the age risk determining the time of occurrence of cases in the family. They state that rheumatic fever does not exhibit the usual characteristics of a communicable disease.

**Age**—The age at which rheumatism starts is about three years, after which it increases very slowly to the age of five. At five the increase is extremely rapid, reaching its maximum about seven and from thence to twelve years there is a steady fall. In both girls and boys therefore, seven is the age at which rheumatism is most prevalent (Fig. 69).

**Environment**—Repeated investigations have shown that there is a very close connexion between dampness and rheumatism. Clay soil which prevents water draining away properly, dwellings near rivers and insanitary houses are all in some way associated with the disease.

**Seasonal variation**—In an analysis of 172 cases in the year 1919-1920<sup>3</sup> it was noted that the majority of the attacks occurred in November and April and that the weather in those two months was extremely inclement, being both cold and wet. Rheumatism is associated rather with the winter than with any particular month.

**Scarlet fever**—In some cases rheumatic carditis may first make its appearance shortly after a severe infection with scarlet fever.

**Strata of society affected by rheumatism**—It is interesting to note the class of person which rheumatism attacks. It is not the wealthy, in fact it is most uncommon in those who are financially well off. Nor is it prevalent among the labouring classes. Rheumatism is a disease of the intellectual poor—the clerk who is ambitious and clever, and inadequately

<sup>1</sup> Schlowger, A., Sherry, A. W., & Payne, J. Further Studies on the Pathology of Acute Rheumatism. *Lancet* May 14 1933, 629.

<sup>2</sup> Schlowger, A., Sherry, A. W., & Payne, J. Further Studies on the Pathology of Acute Rheumatism. *Experimental and Clinical Evidence of a Genetic Susceptibility to Acute Rheumatism*. *Lancet* May 18 1933, 1143.

<sup>3</sup> Eagles, Evans, Fisher, & Keith. A Study in the Etiology of Rheumatic Diseases. *Lancet* Aug. 21 1920, 411.

<sup>4</sup> M. G. Wilson et al. The Familial Etiology of Rheumatic Fever. *Genetic and Epidemiologic Studies*. *Joint Publication*. April and May 1943, 48 & 49.

<sup>5</sup> F. J. Poynton, Donald Paterson and J. C. Spence. "Acute Rheumatism in Children." *Lancet* 1939, 1046.



fed and nourished is the typical victim. Devoting more of his money to appearance and education than to food he forms the ideal soil for the disease, while the labourer who makes no attempt to keep up appearances but feeds himself comparatively well appears to be more or less immune. The fact that it is people with the ambitious outlook of mind who are affected must be taken into account. They are unwilling or too keen on their work to convalesce for the proper period. Gradually, however, the attitude of the public is being influenced by education and it is now less common to find the treatment of rheumatism neglected.

**Geographical incidence.**—Rheumatism is extremely prevalent in the British Isles while in some Continental cities it is practically unknown. In America the incidence is probably nowhere so high as in and about London.

**Pathology.**—The organs attacked most rapidly by rheumatic infection are the heart, its coverings and its valves; the subcutaneous tissue especially round the joints; the synovial membrane; the brain and the tonsils. The

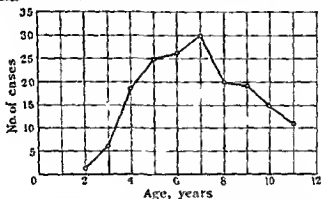


Fig. 69.—Age incidence of initial attack in 172 cases of rheumatism

most characteristic lesions are found in the heart muscle and its valves and in subcutaneous nodules. These consist of a fibrinous area which stains badly and shows no particular structure and a surrounding area containing endothelial cells intermixed with fibroblasts and inflammatory lymphocytes. In the heart muscle itself such nodules are accompanied by large inflammatory cells containing many nuclei. Such an exudate and inflammatory reaction with multinucleated cells is known as an *Aschoff's nodule*.<sup>1</sup> The inflammatory reaction in a valve develops with the lesion. In an early lesion the valve is swollen and there is a tendency to poor staining with lymphocytes and inflammatory cells. Later a fibrinous structureless exudate extrudes on the free surface of the valve. This stains badly, and on the free surface of the exudate the various circulating blood elements tend to deposit. Deep to the fibrinous material, however, endothelial cells, lymphocytes and early fibroblasts appear, so that the essential structure of a damaged valve is identical with that of the subcutaneous nodule or heart nodule. Seldom is one portion of the

<sup>1</sup> V. Coates and C. F. Coombs, "Observations on the Rheumatic Nodule," *Arch. in Child.* 19: 6: 133.

heart alone affected—that is, rheumatic endocarditis is seldom, if ever, present without an affection of the myocardium. Certainly, rheumatic pericarditis does not exist apart from myocardial and endocardial changes. The term *rheumatic carditis* is therefore much to be preferred. Apparently, a widespread tendency to rheumatic "fibrosis" must inevitably produce permanent damage to the organs involved.

### CHOREA

(RHEUMATISM OF THE NERVOUS SYSTEM, RHEUMATIC ENCEPHALITIS)

**Ætiology.**—Chorea or St. Vitus's Dance has been thought by some to be due to fright, by others to shocks or falls, but the more generally accepted theory is that it is a rheumatic encephalitis. A close study of the clinical picture and pathology supports this theory. Air raids were said to have produced much chorea during the 1914-18 war, but an analysis of 84 cases just after the war and also of those occurring throughout the war months, at the Hospital for Sick Children, Great Ormond Street, did not in the least bear this out. Chorea was shown to be seasonal, before, during and after the war, and therefore, if the older theory is to remain tenable, fright would have to be seasonal also.

**Sex.**—Authorities differ whether chorea is twice or three times as common in females as in males, but the preponderance of females is undoubted. No really adequate explanation of this disproportion is offered.

**Pathology.**—The mortality is so low that very little material has been examined *post mortem*. Cases carefully investigated, however, have shown a characteristic cellular infiltration around the small capillaries, comparable up to a point with the picture of encephalitis lethargica. The whole brain is affected, especially the white matter, and in one case the caudate nucleus had suffered grossly, minute emboli and blocking of the vessels by thrombi were also noted.<sup>1</sup> In some cases extravasation of blood from the finest capillaries has been reported.

**Clinical picture.**—Usually chorea occurs at about school age, that is between five and seven. The child is often of the bright, slim, rather nervous type. Some authorities claim that those with Auburn hair are more subject to this infection. The child may flag easily, become unduly emotional, laughing or crying too easily, and lose colour. The tendency to drop or spill things is common, the writing becomes less legible, the speech less articulate. Movements, in standing up or sitting down, are impetuous. The child tends to fall about, catching one foot behind the other, and on this account is often covered with bruises or abrasions, especially about the knees. As the disease develops, all the muscles in the body become involved, even the eye-muscles. The involvement of the facial muscles causes grimaces, so that the child appears to smile or frown for no reason whatever. Constant wriggling movements of the shoulders and involuntary movements of the arms, hands, legs and feet are found. The respiratory muscles are attacked, resulting in inco-ordination between the diaphragm and intercostals, most noticeable when the physician examines the chest, half way through an inspiration

<sup>1</sup> J. G. Greenfield and J. M. Wolfson. "Pathology of Sydenham's Chorea," *Lancet*, vol. 1, 16, 1944, p. 693.

or expiration, there is a sudden antagonism between the two sets of muscles so that respiration may come to a standstill. The knee jerks are active, giving rise to what is known as the 'hung up' knee jerk, that is after the jerk has occurred, the leg fails for a moment to drop back into its previous position. Snorting noises and clucking with the tongue are also characteristic. These constant movements continue throughout the waking period and tend to keep the child from sleep. She becomes worn out and thin and she can only be fed with difficulty. Bursts of crying further depress her. A pronounced case of chorea is a pitiful sight and in some of the more severe cases the child may become almost maniacal. Accompanying most cases there is at least cardiac dilatation, but actual carditis frequently develops. The majority of cases sooner or later develop a heart lesion, in fact, by the second or third attack at least 75 per cent of hearts are affected. The disease is also commonly accompanied by rheumatic nodules but arthritis does not coincide with the chorea. As many as eight or nine attacks have been known.

**Prognosis.**—From the chorea there is very little to be feared that there is no evidence of any permanent cerebral damage and recovery appears to be complete. It is in the accompanying heart disease that the danger lies.

**Treatment.**—There can be little doubt that a child with any rheumatic affection, especially chorea, recovers more quickly when taken out of the environment in which the infection occurred. A dry, warm house and climate are greatly to be desired.

**General management.**—Every patient with chorea ought to be confined to bed during the active stages. Choreiform movements mean that rheumatic infection is present and active and the heart is therefore in danger. The child should be placed flat in bed with a very thin pillow or none at all. Restraint, in the form of shoulder straps should be provided if there is danger of the patient sitting up. The sides of the bed should be padded to prevent banging of the head and limbs. The pulse is much more important than the temperature, in that it furnishes an indication of the cardiac condition, this is particularly true of the pulse-rate during sleep.

**Nursing.**—Much skill is required in the management of chorea patients. They must very often be fed from a feeding cup, and in the more severe cases should never be allowed to feed themselves. They require great tact in their management, as with their unstable emotions any attempt at discipline must be discontinued.

**Diet.**—The maximum amount of food which they are able to assimilate without overloading the intestinal tract should be given, but it is advisable to confine feeding to three meals per day. Not much fatty food should be given, and a diet rich in milk, cream and eggs is to be avoided. For a specimen diet sheet, see p. 17.

**Drugs.**—A great variety of drugs has been employed. Aspirin or sodium salicylate may be given during the acute stages—rather on general lines as they do not appear to have any specific effect on the chorea. Calcium aspirin, 3 to 5 grams three times daily, may be continued safely for some weeks. Sedative drugs are best used in the chronic stages. To

understand the drug treatment of chorea it must be realized that there is an organic and a functional element. During the acute stages the disease is almost entirely organic. In six or eight weeks, however, provided the pulse has settled and the condition of the heart is satisfactory, it may be safely assumed that the movements are largely functional. Amytal, (Lil Lilly)  $\frac{3}{4}$  grain with aspirin, 5 grains, may be given twice or three times daily. The movements are lessened at once. Amytal can be obtained in  $\frac{1}{4}$  grain and  $\frac{3}{4}$  grain tablets separately, if aspirin is not required. Luminal sodium ( $\frac{1}{2}$  grain) together with a little bromide (1 grain), made up to 2 teaspoonful with chloroform water and given night and morning is a good substitute.

Re education—During the chronic stage the functional element is

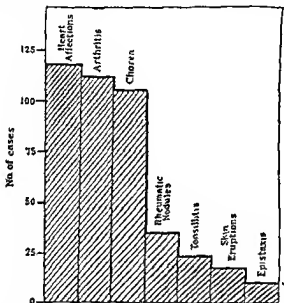


Fig. 70 Table showing the relative incidence of symptoms in 172 cases of rheumatism in childhood (Lancet)

greatly improved by attempts at re educating the muscles. If the patient is given such occupations as knitting and sewing walking along a mark on the floor, or feeding herself improvement of the finer movements of co ordination is encouraged.

Removal of tonsils—In all cases where the tonsils are enlarged and septic they ought to be removed.

#### OTHER RHEUMATIC MANIFESTATIONS

In addition to chorea rheumatism is apt to attack the heart (carditis) the joints (arthritis) the muscles (myositis) the subcutaneous tissues (nodules) the throat (tonsillitis) and the skin (rheumatic erythema and purpura) (Fig. 70).

**Clinical picture.**—The general appearance to which advancing rheumatism gives rise is the same whatever the system attacked. The child flags, becomes pale and listless, loses weight and appears anæmic. There is complaint of pains in the limbs, down the thighs in the bones and in the shins, these are termed *growing pains*. The pains are often also present in the shoulders and arms in the back, and in the spine at the back of the neck. Stiff neck and crick in the neck are common and some children may get a persistent stitch in the side which it is suggested, is really an early muscular infection. As a rule there are recurrent attacks of sore throat with enlargement of the tonsillar glands though the enlargement may not be noticeable. There may have been a sharp attack of scarlet fever, or the child may have complained of breathlessness and palpitation after exertion. Such symptoms as those described are seldom found in the well nourished—almost invariably the children are much under weight. A dirty tongue is a constant accompaniment of active rheumatism.



Fig. 71.—Rheumatic nodules on knees

**Nodules**—These are looked for over the patellæ, the elbows and the back of the skull, also over the tips of the spines of the vertebree on the knuckles and the ankles, and on the spines of the scapula. On bending the knee, elbow, fist or ankle slightly, so as to draw the skin tensely over the bony prominences, the nodules stand out as white marks (Fig. 71). Pressed on slightly, they slip and slide beneath the finger. Examination of an excised nodule shows a localized inflammatory focus. Nodules may appear extremely rapidly and disappear just as rapidly, even within a few days a well marked nodule may be gone. They are a sign of extremely active rheumatic infection and are generally accompanied by a gross cardiac lesion. They have been noted, however, where very little carditis appears to be present. Of 33 cases with nodules noted by the author, 37 per cent. were fatal and 17 per cent. had very severe heart disease. In only 2 cases was the heart free.

**Arthritis**—Unlike the picture in adults, very little is seen on examination of the affected joints in children. There is seldom any swelling, redness or heat although the pain may be excruciating and the child may resist the slightest movement. Occasionally, however, the adult type

with swelling is found. In the more severe infections accompanying arthritis there is involvement of the vertebral articulations in the neck and back. In such a case the patient is unable to move the head in any direction and a mistaken diagnosis of meningitis has been made. Arthritis is much commoner in boys than in girls, possibly two boys to one girl, and in this way the disproportion in the sexes seen in chorea is equalized. As a rule, an attack of tonsillitis precedes, or is coincident with, an attack of arthritis.

**Tonsillitis**—So often does tonsillitis accompany or precede rheumatism that it is reasonable to believe that the tonsils are the portal through which infection enters the body. The appearance of an acute rheumatic tonsillitis is characteristic, there being a thin, pale exudate on the tonsil, as if the child had been recently drinking skimmed milk.

**The skin**—Two types of rash are noted. (1) *rheumatic erythema* or erythema marginata, which appears over the chest and back, occupying a great area, the edge is characteristically serpiginous or map-like, changing from hour to hour. Such a rash tends to come and go for several weeks in some cases, it has been called erythema annulare rheumaticum. (2) *rheumatic purpura*, which is seldom seen except in malignant or fatal cases where the cardiac vegetations are luxuriant. As a rule, these cases are fatal.

### RHEUMATIC CARDITIS<sup>1</sup>

The whole significance of rheumatism and the diagnosis in the majority of cases depends upon the extent to which the heart is implicated. Vague limb-pains without cardiac involvement may or may not mean rheumatism; with cardiac involvement the diagnosis is confirmed. When the heart is involved to a minor degree it is merely dilated, this is often seen early in cases of chorea and arthritis. On examination, the apex is found to be slightly outside the nipple line, and on auscultation at the apex there is a very soft systolic murmur, probably due to regurgitation at the mitral valve from a dilatation of the mitral ring. With a more severe and prolonged infection in addition to dilatation the valves are involved, the mitral valve being by far the most commonly affected, with regurgitation. During the healing of severe mitral involvement, stenosis almost invariably occurs. Mitral stenosis and regurgitation is the end result of a vast proportion of rheumatic cardiac infections. In *mitral regurgitation* the heart is found to be enlarged to the left, due to hypertrophy of the left auricle and (to a lesser degree) of the left ventricle. No thrill is present over the præcordia. On auscultation a systolic murmur, propagated into the axilla, is noted. This murmur can also be traced backward to the level of or beyond the nipple-line. Where *mitral stenosis* and regurgitation co-exist, there is also enlargement of the heart to the left, but in addition there is a distinct thrill over the præcordia. If the heart is acting well the first sound is obliterated by the pre-systolic and systolic murmurs and the second sound is short and sharp. Where the heart works less perfectly and compensation is not so good, the pre-systolic murmur breaks off and moves forward so that it becomes mid diastolic. This may be

<sup>1</sup> A. Wallgren, "Studies on Erythema Annulare Rheumaticum," *Acta Paed.* 1935 xvii, Fasc. 4.

<sup>2</sup> E. C. R. Cooper, "Rheumatic Infection in Childhood," *Arch. Dis. Child.*, June, 1913, xviii 88.

imitated by the sounds "too ti froo" When compensation and function are still less perfect, this presystolic murmur may become actually diastolic. In this case it imitates the sound made by bellows and is known as a "bellows murmur". The blast outward of the bellows represents the systolic and the withdrawing of air the diastolic murmur.

The aortic valve is the next most commonly attacked. Regurgitation results, but, as a rule, not until after the mitral involvement. In the aortic area of the child (which lies to the left and not to the right of the sternum as in adults), the second sound is replaced by a soft murmur, the heart as a rule showing hypertrophy of the left ventricle. Aortic stenosis is still less common.

Tricuspid involvement is extremely rare, and pulmonary involvement practically unknown.

Where there is an active rheumatic infection in the heart the pulse<sup>1</sup> is almost always increased in rate, this being so universal that the pulse rate, especially during sleep, is of extreme importance and ought to be firmly relied upon. The charting of pulse-rates is much more helpful than the temperature.

#### RHEUMATIC PERICARDITIS

Like acute tonsillitis, rheumatic pericarditis is remarkable in that it gives rise to a high temperature. Most rheumatic infections are not accompanied by fever, especially if salicylates are being given. Despite salicylate therapy, however, pericarditis runs a high fever for some days or even weeks, the child seeming greatly distressed and having a pinched appearance. There is dyspnoea and often a short hacking cough reminiscent of pneumonia. There is often a scattered bronchitis, but at the left base, in almost every case, at the angle of the scapula, will be found a patch of high pitched tubular breathing. The cause of this is debatable, some regarding it as a rheumatic pneumonia and others attributing it to compression of the left bronchus by the enlarged heart and a consequent collapse. The heart is enlarged and the pulse rapid. On auscultation a harsh to and fro murmur, most audible over the sternum, is heard. Seldom is there much fluid, consequently, the heart sounds are not damped down or distant. Actually, if examined *post mortem*, both the parietal and visceral surfaces of the pericardial sac are covered with a thick, felted coating, which in many places is adherent, with possibly a very small quantity of opalescent fluid lying in loculated areas.

As in other rheumatic infections, correct nursing is essential, the child being kept very flat and still. An ice-bag is helpful in many cases of precordial pain. Digitalis therapy should be tried, although in the presence of fever it is often unavailing (see p. 167).

Pericarditis may complicate both chorea and arthritis and is probably the most serious cardiac lesion, tending to leave the pericardium adherent and thus gravely disabling the heart.

#### ADHERENT PERICARDIUM

In an effort to compensate for this disability the heart hypertrophies to a great extent, and in cases of rheumatic carditis the *cor bicornium* is

<sup>1</sup> D. Schlessinger "A Study of the Sleep Pulse-rate in Rheumatic Children." *Quart Jour Med.*, New series, No. 1, Jan. 1927, 1-67.

frequently seen *post mortem*. Clinically, adherent pericardium may be suspected from the size of the heart and the preceding pericarditis but, in addition, inspection of the intercostal spaces in the left axilla and at the angle of the scapula will often show a dragging in and out of the chest wall with each cardiac impulse. The outlook in cases of adherent pericardium is extremely grave, most of the patients ultimately becoming hopelessly crippled or dying early.

**Care of a rheumatic child with involvement of the heart.**—As in the typical case of chorea described on p 856 the child should be kept lying perfectly flat and if necessary, restrained in this position. The pulse should be charted four hourly or at least twice daily (Fig 72). Sodium salicylate should be given, with bicarbonate of soda, in doses of 20-40 grains daily or aspirin or calcium aspirin, 5 grains three or four times daily according to age.

The diet should be as in chorea, light and nourishing yet one not likely to make the child bilious. It should be pushed just short of the limit of digestion.

If the pulse rate drops rapidly with rest, there are no murmurs, and the dilatation of the heart rights itself a little more strain may be thrown on the heart. A second pillow, then a third, then a back rest may be allowed. Later the child should sit up and feed himself, and finally may get up and slowly resume his ordinary life. The pulse-rate, however, is the all important factor in controlling this resumption. At any sign of a rise of pulse rate or of the appearance of a murmur or fresh cardiac damage, the child should be made to revert to the flat position. This state of absolute rest may have to be maintained for many weeks if the cardiac damage is considerable. The parents should be instructed to take the child's weight at not longer than monthly intervals, and at any signs of loss, fresh limb pains, increased shortness of breath, or pallor, a recurrence may be expected and medical aid should be sought. Of recent years the sedimentation rate has been determined in suspected exacerbations of rheumatism, and constitutes a most helpful and reliable guide to the progress of the disease. To get the child well nourished and healthy and keep him so is the only safeguard against a future attack.

**Sedimentation rate in rheumatism.**<sup>1</sup>—The sedimentation test is performed by allowing citrated blood to fall in a special capillary tube for a period of one hour, the distance fallen by the red cells being then measured. In health the fall is less than 10 mm., but in disease it is greater. Acute infections, such as tonsillitis and influenza, cause a very great and rapid fall and in rheumatism it may reach to 50 mm. in the hour. The rapidity of the fall has been found an excellent guide to the activity of the rheumatic lesion. In chorea there is no increase in the sedimentation rate. In cardiac failure the rate may be normal although clinically the child is rapidly deteriorating. In rheumatic arthritis the author has found the rate much raised. It is, however, raised also in anaemia, and a correction must then be made for the anaemic state of the blood in all cases of infection.

<sup>1</sup> W. W. Layne and Bernard Schlesinger, "A Study of the Sedimentation Rate in Juvenile Rheumatism," *Arch. for Child*, No. 60, Dec 1935, p. 403.



**After-care.**—Open air schools and prolonged convalescence under the care of persons who understand the problem should be advocated. Septic teeth and tonsils should be dealt with in every case as soon as the active signs of rheumatism have sufficiently subsided. Just as in adults the

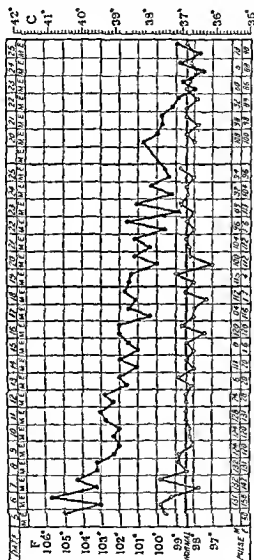


Fig. 72.—Temperature and pulse chart in a case of rheumatic carditis and the lower line the temperature and the upper line shows the pulse rate

gauge of what a child should be allowed to do is an individual one. By slow degrees, more and more strain may be thrown on the heart. If the child continues to gain weight, and his cardiac compensation continues to be satisfactory, there can be no harm in letting him resume a normal life, but at any sign of a breakdown in either, retrenchment must be

imposed and a resumption made on much slower and more cautious lines.

**Prognosis**—With efficient care the mortality is extremely low in all types of rheumatism probably not exceeding 5 to 10 per cent. The proportion of complete recoveries is about 50 per cent, the remainder are either moderately or grossly disabled. If treatment is not followed up the mortality in any given group of cases rises steeply almost all the severely damaged or untreated cases succumbing at an early age.

Among the more severe cases of carditis the prognosis is gloomy as shown by Cotton (1912)<sup>1</sup>. Among 200 boys whose average age was 11 years and who were observed over a 10 year period slightly more than one half were alive and more than one third were dead the remainder being traced. The death rate in children with mitral stenosis and aortic regurgitation was much higher than in those with mitral systolic murmurs and children with moderate or considerable enlargement of the heart are less likely to live 10 years than those with slight or no enlargement.

### How to prevent Rheumatism

The most effective method of prevention is to keep the child thoroughly fit. If he has bad teeth or septic tonsils they should be attended to early so that he is never allowed to get run down. He should be weighed at intervals and at any failure to gain weight over a considerable period he should be examined carefully for organic disease and his diet and home regime carefully investigated to ascertain the error. Damp districts and damp houses should be avoided and the child must not be allowed to put on or wear damp underclothing after he has exerted himself and perspired. After games he should be given an opportunity to change his clothes completely especially the underclothes. His diet should be a general all round well balanced one<sup>2</sup> containing all the food elements including a pint of milk daily and a liberal allowance of the various vitamins.

At any sign of growing pains, pallor or anæmia, lassitude, sore throat, undue nervousness or emotional instability he should be carefully examined and steps taken to deal with such symptoms as they may be the earliest manifestations of rheumatism.

### RHEUMATOID ARTHRITIS<sup>3</sup>

(Still's Disease)

This condition in childhood was very fully described by Dr G. F. Still in 1897<sup>4</sup> and very little has been added to his original description.

**Ætiology**—The affection is commoner in girls than in boys and the majority of cases commences in the second, third or fourth year of life. The cause is not definitely known but is usually thought to be due to an infection gaining entrance either through the tonsils or from some septic

<sup>1</sup> T. F. Cotton, *Some Aspects of Carditis*, *Brit. Med. J.* Nov. Oct. 21st, 1912, II, 473.

<sup>2</sup> A. P. Thompson, *The Effect of Variation of the Diet in Rheumatic Children*, *Arch. Dis. Child.* No. 1, 1919, IV, 15.

<sup>3</sup> F. J. Paynton in Garrod, Batten, Turnbull and Paterson's *Diseases of Children* (1933), p. 261. H. Rolleston and H. A. T. Fairbank *ibid.*, p. 262.

<sup>4</sup> G. F. Still, *Med. Chir. Trans.* London 189, lxxx, 47.

focus in the sinuses or elsewhere. Bacteriological examination of the periarticular tissues and of any effusion into the joints has repeatedly shown these to be sterile.

**Pathology.**—The chief changes are periarticular and the joints most commonly involved are the knees ankles wrists elbows and interphalangeal joints. Every joint in the body however may be involved and commonly the vertebrae are grossly affected. With disuse and the accompanying general ill health there is marked osteoporosis of all the bones, revealed by X ray examination. There is general enlargement of the glands, the epitrochlear glands being invariably swollen and the inguinal and axillary glands also being prominent. The spleen is occasionally involved. In a case of long standing the synovial membrane is thick and gelatinous and the cartilage becomes more and more eroded, until finally it shows gross destruction, with partial or complete ankylosis of the joint.

**Clinical picture.**—A child aged two or three has an acute feverish illness, the joints rapidly becoming swollen and very tender. Fever is not always prominent however, and may escape notice. Movement is limited at the elbows wrists fingers, knees and ankles. The head is moved with difficulty, and chewing may be impeded because of pain or stiffness in the temporo maxillary joints. The appearance of the various joints is typical—a fusiform swelling about the joint with wasting of the muscles above and below (fig 73).



Fig 73 Rheumatoid arthritis. Note obvious involvement of the knees ankles and wrists.

Although pain is often a feature at the very onset, the joints rapidly become much less painful finally becoming practically painless unless overmanipulated. At intervals there is an acute fever for a day or two, with malaise and a return of pain to the joints, at the same time the glands become slightly more enlarged and tender, and then again subside. The spleen is usually palpable. The sedimentation rate is greatly raised in this condition, a rate of fall of 40 to 60 mm in the hour being not uncommon. It is likely to remain high for a longer period.

**Differential diagnosis.**—Because of the stiffness of the neck, spinal caries must be ruled out by X ray examination. The chronic joint involvement excludes acute rheumatism, and this is supported by the fact that

the heart is not involved Syphilitic arthritis, as a rule, involves the knees only and the Wassermann reaction serves to exclude syphilis

**Prognosis.**<sup>1</sup>—Colver, in a recent survey of 69 cases comes to the conclusion that the disease is self limiting, lasting in all about five years, below the age of 5 years the mortality is 30 per cent, above this age the mortality is very low Complete recovery takes place in one case in four, and is limited to those in which recovery took place within three years Very gross crippling is uncommon Surviving patients are usually capable of earning a livelihood

**Treatment.**—The removal of septic foci, such as infected teeth, tonsils or sinuses, should first be undertaken Local treatment to the joints, such as massage passive movements, artificial sunlight and radiant heat, is very useful Cases of complete cure have been reported after acute infectious diseases such as scarlet fever and septicæmias, especially streptococcal septicæmias In one case, after tonsillectomy the child developed a septicæmia, and when this subsided all signs of the rheumatoid arthritis rapidly disappeared The child was seen one year later and appeared perfectly healthy This suggests some form of protein shock or anaphylaxis In the author's experience more hope lies in treatment along such lines than in any other direction Protein shock,<sup>2</sup> induced with T.A.B. (typhoid, paratyphoid A and B) vaccine, sterile milk, or peptone injections given intravenously has been employed but has not been successful in the author's hands

**Gold<sup>3</sup> therapy**—Recently the author has been using myocrisin (May and Baker) or sodium aurothiomalate with some success, the dosage being 25 mgm per week for 6 or 8 doses After an interval of 1 or 2 months, a second course may be given, depending on the sedimentation rate, condition of the urine and general clinical progress The urine should be examined from time to time and occasional blood-counts done, and frequent sedimentation rates are necessary to note progress It is usual for the child to react with some rise of temperature

Where gross deformity is present, orthopædic treatment must be undertaken Special boots and irons prevent further contractions and deformity.

No special diet is required, but tonics, such as cod liver oil and iron, are indicated

<sup>1</sup>T Colver "The Prognosis in Rheumatoid Arthritis in Childhood" *Arch. Dis. Child.*, Aug 1923 xii, 252.

<sup>2</sup>J H Thurnfield "Notes on a Case of Chronic Polyarthritis (Still a Disease) Treated by Intravenous Protein Therapy" *Arch. Dis. Child.*, 1927 ii 173

<sup>3</sup>M B Ray "Note on the Treatment of Chronic Arthritis with Calcium Aurothiomalate," *Practitioner* Jan. 1943 xlii 49

## CHAPTER XX

### ENDOCRINE DYSFUNCTION IN CHILDHOOD

#### PITUITARY DISORDERS

(1) **Pituitary gigantism.**—This is a rare condition in childhood, although it may be seen more commonly in adults as acromegaly. Hypersecretion of the anterior lobe of the pituitary gland causes an excessive growth of the bony skeleton and the viscera. Over action of the anterior portion of the pituitary may be caused by an adenomatous growth of the chromophil cells. An X ray of the pituitary fossa may show an enlargement and help to establish the diagnosis.

Gardiner Hill<sup>1</sup> states that, for true pathological gigantism, an excessive activity of the anterior lobe chromophil cells of the pituitary during the active growth period is necessary. He classifies gigantism into (A) the simple hereditary type, and (B) the endocrine group. In the hereditary type this undue stimulus appears to run in families. The endocrine group he subdivides further into (a) hyperpituitary gigantism where the growth and differential growth curves show an exaggeration of the normal (general symmetrical over growth is the characteristic feature, though in some instances localized overgrowth in the form of acromegalic changes may be in evidence too) (b) hypogonadal (decrease of sex gland activity) gigantism where an additional feature favouring overgrowth is present that is, delayed closure of the epiphyses. In these individuals the growth curve in adolescence is prolonged beyond the normal and often far into adult life.

(2) **Pituitary obesity** (Fröhlich's syndrome).—This rare condition should not be confused with the obese child (see p. 18). In Fröhlich's syndrome, which is due to a tumour of the pituitary, or of failure to function the child is stunted and fat. The male shows a feminine distribution of fat and the female a marked exaggeration of the normal. The child is slow mentally and may show symptoms such as blindness due to the tumour. There is absence of hair on the body, and delay in the secondary sexual characteristics. The genitals show marked hypoplasia and the carbohydrate tolerance is much increased. Large quantities of glucose can be taken by the mouth without appearing in the urine.

(3) **Diabetes insipidus**.—This is a rare condition which may be due to a pituitary lesion or to a lesion of the hypothalamus. It may be caused by congenital syphilis, encephalitis, tumour, cerebral malaria, trauma, actinomycosis, xanthomatosis, leukaemia, Hodgkin's disease, Boeck's sarcoidosis, or pellagra. Occasionally the disease is hereditary.

**Clinical picture.**—The child remains small and stunted. There is great thirst, and much urine is passed. Several pints may be drunk, and passed, each day. The urine is pale and of low specific gravity but is otherwise normal.

**Treatment.**—A recent case under the author's care, published by Court and Taylor, 1933<sup>2</sup> was completely controlled by two injections per week of concentrated emulsified pituitary of 1 c.cm., each.<sup>3</sup>

(4) **Progeria.**—This is an excessively rare condition in which the children at or shortly after birth, have the appearance of senility, and are carried off by intercurrent diseases. The condition was named by Hastings Gilford. The children are small

<sup>1</sup> H. Gardiner-Hill, "Abnormalities of Growth and Development (Clinical and Pathological Aspects)", *Proc. Med. Soc.*, June 1937, 1 p. 11.

<sup>2</sup> H. G. Wylie, "Diabetes Insipidus, its Clinical Features and Treatment", *Proc. Roy. Soc. Med.*, 1933, 26, 351.

<sup>3</sup> D. Court and S. A. Taylor, *Proc. Roy. Soc. Med. (Children's Section)*, July, 1933, 26, 1293.

hairless, wrinkled and old. It appears to be due to a deficient secretion of the anterior pituitary from birth.

## THYROID DISORDERS

(a) **Cretinism**—This condition may be present from birth or acquired at some later date. In the British Isles it is as a rule congenital.

### CONGENITAL CRETINISM

Diagnosis is possible at the age of 3 to 4 months. The sex distribution is equal.

**Ætiology and pathology**—No explanation has been offered of the



Fig. 74.—Showing state of thyroid after death in case of cretinism.

A. Larynx with no trace of thyroid.

B. Larynx of a normal child showing well marked thyroid.

congenital absence of the thyroid gland and it may be looked on as a malformation. The absence in most cases is complete (Fig. 74).

Absence of the thyroid gland produces a typical clinical picture and certain pathological sequelæ throughout the body.

**Clinical picture**—The cretin is pale and has a yellow tinged, harsh skin. The hair is straight, dry and sparse, the voice is hoarse and low-pitched, the tongue is greatly enlarged, and protrudes from the mouth continuously, since it is actually too large for the mouth to contain (Fig.



Plate 22 —Radiogram showing syphilitic epiphysitis  
Note also the well marked periostitis

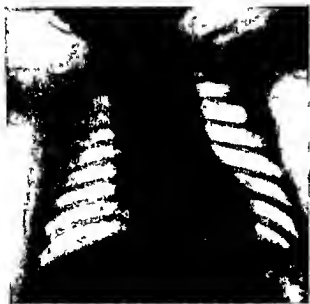


Plate 23 — Radiogram showing enlarged thymus in an infant ten weeks old



75) The body is firm and stiff and inclined to be muscular. The child is slow in all his movements, the smile comes and goes slowly, and the mentality is greatly retarded. He is very backward in learning to walk, and may be several years old before he can accomplish this, if untreated. He is inclined to make snorting noises with his nose. The temperature is subnormal, the hands and feet being cold, and the pulse slow. He is chronically constipated. The growth of the skeleton is slow and he is dwarfed in size. An X ray of the wrist will show marked delay in the appearance of the centres of ossification. His teeth are late in appearing and when they do appear, tend to be carious. The thyroid gland cannot be felt, but it is seldom that cretinism is accompanied by other forms of congenital malformation. The abdomen is large, and a well marked umbilical hernia develops.

An excess of fat accumulates in the subcutaneous tissues, especially about the shoulders, just as in the hibernating bear, fat is also laid down in many other parts of the body, such as the tongue and the subcutaneous tissues.

Very often, mistakes are made in diagnosis between cretinism and mongolism. There ought to be no difficulty whatever, as the following summary will show.

#### CRETIN

Pale, yellowish skin.  
Skin harsh.  
Hair straight, dry and sparse.  
Tongue greatly enlarged and protruding continuously.  
Voice harsh and low pitched.  
Body firm and stiff, inclined to be muscular.



Fig. 75.—An untreated cretin aged 5 years, showing the large abdomen with umbilical hernia, protruding tongue, and pads of fat on the shoulders.

#### MONGOL

Pink, cheeks and fresh looking.  
Skin soft.  
Hair quite normal.  
Tongue small, pointed at the tip, protruded at times with a sucking movement.  
Normal cry.  
Body soft, hypotonic and flabby.

Movements slow

Backward in learning to walk

Snorting noises in nose

Temperature subnormal

Chronically constipated

Growth of skeleton slow dwarfed, teeth  
ing backward

Eyes normal

Thyroid gland cannot be felt

Circumference of head normal

Seldom accompanied by congenital mal-  
formations

Movements quick

Normal or only slightly backward.

Snorting noises in nose

Temperature normal

Not necessarily constipated

Not necessarily under sized with proper  
diet teething normal

Eyes slanting and almond shaped

Thyroid gland palpable

Greater or lesser degree of microcephaly

Heart disease or some other malformation  
often present



Fig. 76.—Cretin before and after several years' thyroid treatment

**Prognosis**—In cretinism this is a matter of great difficulty, as it depends on how early and how efficiently the child is treated. There are, however, a few cases in which there is mental deficiency despite treatment with thyroid. The majority of cretins are treated late and inefficiently, and therefore lag some distance behind the normal child, failing to catch up or reach the normal standard. A small proportion, taken in hand early and properly treated, become apparently absolutely normal.

**Treatment**—Thyroid gland must be administered, beginning with small doses. The author uses Thyroidinum vaccin B. P., and, for an infant a few months old, begins with a dose of  $\frac{1}{16}$  grain three times daily, slowly increasing this week by week until the greatest dose is reached which the infant can tolerate without diarrhoea and without undue loss of weight. This is usually about  $\frac{1}{2}$  grain in the day. The signs of recovery are that the constipation ceases, the cold hands become warm, the dry hair soft and even curly, the increased subcutaneous fat disappears, the skin is

soft and loses its yellow tinge becoming pink and white. The rate of the heart's action should be normal for the child's age. The voice ceases to be hoarse and is clear and infantile. The tongue is small and does not protrude from the mouth, the mentality is quickened and the child smiles and obviously appreciates its surroundings (Fig. 76).

**Signs of too much thyroid**—When too much thyroid is being administered there is a tendency to undue loss of weight, looseness of the bowels and an elevated temperature. The child is nervous and fearful, being too timid to cross the street and afraid of cats and dogs. Finally there are attacks of palpitation with sudden fainting fits probably accompanying the palpitation. An average cretin a year old will tolerate



Fig. 77.—Goitre in a child who later became a typical cretin. On treatment with thyroid the child became normal but the enlarged thyroid persisted.

about 1½ grams in the day, and at between one and four years appears to tolerate 2 to 3 grams per day. After that the dose remains stationary. Each child, however, is a law unto itself, and only by very careful observation can the proper dose be ascertained. The dose of thyroid can be controlled by metabolic estimations, especially in older children.<sup>1</sup> During the winter months the dose of thyroid is a larger one than during the summer. For instance, a child who tolerates 3 grams during the winter months will tolerate not more than 2 grams during July and August.

After the administration of thyroid there is a rapid mental and physical

<sup>1</sup>H. Gardiner-Smith, J. G. Brett and J. Forrest Smith, Carbohydrate Tolerance in Myxedema, *Quart. Jour. Med.*, No. 71, April, 1923, xviii, 327.

development and a greatly increased growth of the bones, which brings in its train the danger of rickets. It is essential, then, that the diet should be carefully investigated and adjusted, and that cod liver oil should be administered at all times along with the thyroid.

#### ACQUIRED CRETINISM

Occasionally an infant is born normal and in the first few years of life has some acute illness or local inflammation in the region of the thyroid. Typical myxœdema follows and cretinism results. Such cases react rapidly to treatment with thyroid. Adenoma of the thyroid, or benign goitre, is very rare in the British Isles.

(b) **Puberty goitre.**—It is common at puberty, especially in girls, to find some enlargement of the thyroid gland with mild symptoms of hyperthyroidism. At the onset of each menstrual period, the neck enlarges and the child tends to be emotionally unstable, and to flush unduly easily. No treatment is necessary, and with the establishment of puberty the condition seems to right itself.



Fig. 78.—Exophthalmic goitre probably congenital. The upper figure shows the child aged 6 months and the lower one aged 9 years.

(c) **Endemic goitre (Derbyshire neck).**—This is an extremely rare condition in the British Isles although cases have been reported especially from Derbyshire and Gloucestershire. It is said that a deficiency of iodine in the soil and water is a contributing factor, although others state that an infection with bacteria or parasites is the cause.

Iodized salt used with the food is said to control the disease.

In such cases, the thyroid may undergo cystic or adenomatous changes, and in a proportion of cases the child may show signs of thyroid insufficiency.

(d) **Thyrotoxicosis (Graves' disease, exophthalmic goitre, hyperthyroidism).**—This is extremely rare in childhood. As puberty

approaches it becomes more frequent. Females are affected twice as often as males.

**Clinical picture**—This is similar to that seen in adults, namely, flushing, tachycardia, sweating and exophthalmos. The appetite may be large and there is a loss of weight.

**Ætiology**—This is not known, but in a proportion of cases the condition appears to be hereditary.

**Treatment**—Five minims of Lugol's solution (iodine in potassium iodide), three times daily, given in milk, a week off and a week on is recommended. In the author's experience surgical treatment appears to be most successful, and children stand operation very well. Complete recovery appears to be possible after excision of part of the gland.

## ADRENALS

(a) **Sarcoma of cortex (Infant Hercules).**—In this condition a malignant growth of the suprarenal cortex causes signs of precocious puberty, and overgrowth of the musculature of the body. It is more often found in girls than in boys, but is extremely rare.<sup>1</sup>

(b) **Sympathoblastoma or neuroblastoma of medulla.**—A tumour composed of nervous tissue is found in connection with the medulla of the suprarenal or of the abdominal sympathetic. Two types are described.

**Pepper type**—In this syndrome the tumours show metastases chiefly in the liver. Occasionally, the tumour appears to be congenital.

**Hutchison type**—In this syndrome the tumour shows metastases chiefly in the skull causing protrusion of the eyes and malformation of the skull bones. Metastases are also found in the ribs, bones of the extremities and liver and kidney.

**Clinical picture**—In a series of 25 cases over a period of 15 years at Great Ormond Street, described by Ruby Stern and G. H. Newns<sup>2</sup> the children complained of the following symptoms: enlargement of the abdomen, pains in the limbs, pallor, wasting and listlessness, and swelling of the head. Proptosis of the eyes, and enlargement of the liver was common, and in three-quarters of the cases some fever was present. Anæmia of the orthochromic type was found. X rays of the skull and long bones showed nothing at the site of the tumour.

**Sex.**—Males were twice as common as females. The average age of the Hutchison type was 3½ years, and of the Pepper type, 2½ years.

**Pathology**—The tumours form large masses in the abdomen, usually retroperitoneal, and sometimes arise from the abdominal sympathetic. Metastases are found in the kidney, lung, pancreas, intestine, ovary and brain as well as in the liver and bones and they arise equally commonly from both suprarenals.

<sup>1</sup> A full description of this and of suprarenal carcinoma is to be found in *Diurnal Latten & Thursfield's Diseases of Children*, 3rd Edn. (Arnold) by E. A. Cockayne pp. 551 and 552.

<sup>2</sup> *Arch. Dis. Child.* No. 71, Oct. 1932, xlv, 267.

**Diagnosis**—Wilms' embryoma does not show the early metastases which this tumour does.

**Treatment**—No treatment is of any avail.

## GONADS

### PRECOXIOUS PUBERTY (MACROSOMIA PRÆCOX)

In this condition there is precocious development of the reproductive organs, and such children appear remarkably overgrown. They do not, however, exhibit gigantism in adult life, in fact in some cases they are unusually small. Premature closure of the epiphyses associated with early maturation of the reproductive system, results in cessation of longitudinal growth before the usual age. The cases of precocious puberty fall into three classes—

(a) *Tumours of the sex glands*—In the female this is found with granulosa-cell tumours of the ovary. Such tumours secrete large quantities of oestrogenic substances, which are responsible for the sex changes and over accentuation of the female sex characters. Menstrual and mammary development may appear from one year onwards. In the male precocity occurs from testicular tumours, particularly of the interstitial tissue and it is these interstitial cells which secrete the testicular hormone, which influences the development of the secondary sexual characteristics.

(b) *The pincal syndrome*—This is similar to the previous one, and has been said to be due to pincal tumours in boys. It is now thought to be due to a destructive process in the walls of the third ventricle.

(c) *The juvenile adreno-genital syndrome*—This chiefly affects females, and is produced by tumours of the adrenal cortex, either benign or malignant. In the female, secondary sex characteristics are of the male type. Muscular development is a feature, and they are often termed infant Hercules. The essential abnormality in cases of precocious puberty is a disturbance of the time factor of growth and development, and changes which should occur to adolescence are projected into early life.

### UNDESCENDED TESTES

The vast majority of undescended testes right themselves without any surgical treatment or hormonal aid, at or about puberty. The two exceptions are—

1 When the testicle is retained or held up by a hernia. Under these circumstances operation is necessary.

2 Where the child is backward physically, or abnormal in some respect. In such cases Pregnyl Gonad or Antutrin S stimulates the development of the sex glands with descent of the testicles. From 500 to 1,000 rat units per week, up to about 35,000 units are necessary in some cases.

### THYMUS

The thymus gland gets gradually larger from birth up to the age of puberty, and then regresses. Various symptoms have been attributed to its enlargement. Among these symptoms are sudden death, thymic asthma, collapse and fainting, and breath holding attacks.

Occasionally an infant is seen who gets sudden attacks of collapse, or who faints when being bathed, and whose X ray shows what appears to be an enlarged thymus. Such a case appears to do very well when radium is applied over the sternum, and a subsequent X ray shows that the thymus has become much smaller. (Plate 23, facing p. 380.)

Occasionally a much enlarged thymus is seen in a child who is wheezing and has obvious respiratory distress. In such a case it is well to apply a tuberculin patch test as tuberculous infection of the thymus is a possibility. Abscess formation in the thymus from a pyogenic organism must also be excluded by Wassermann.

\* A report on 25 treated cases, *Lancet* May 4, 1928, I, 922.

T. W. Allmop, "The Treatment of Imperfect Descent of the Testes with Gonadotropin Hormone," *Lancet* Feb. 27, 1927, I, 487.

Pearce Williams, "Prognosis in Undescended Testicle," *Lancet* Oct. 17, 1926, II, 919.

and leukaemia by a blood-count. Actual tumour formation in the thymus should also be kept in mind (thymoma). Where infection can be excluded, an application of radium should be given, or alternatively, X rays.

#### STATUS LYMPHATICUS, STATUS THYMICO LYMPHATICUS, LYMPHATISM

There is a great diversity of opinion on sudden death due to so called lymphatism. It appears, however, that children with a large thymus, and an excess of lymphatic tissue elsewhere throughout the body, are peculiarly liable to sudden death from slight causes. At operation, and after trivial accidents, they may die suddenly. An excellent summary of the present knowledge of this subject has been made by J. F. Taylor.<sup>1</sup>

#### DWARFISM AND INFANTILISM

Usually, dwarfism and infantilism are found combined, but occasionally dwarfism is found separately. In dwarfism the defect is skeletal, and limited to the skeleton. In infantilism the physical and psychological attributes of childhood persist into adult life. smallness of stature, under development of the musculature, a large head in proportion to the body, comparatively short limbs, lack of function of the genitalia and certain qualities in the mental sphere.

A convenient classification of dwarfism is as follows:—

(1) *Simple dwarfism*—(a) Hereditary, (b) due to developmental skeletal disease such as achondroplasia and dyschondroplasia, and osteogenesis imperfecta, (c) due to acquired skeletal disease, such as rickets, spinal caries and spinal deformities, and (d) hypergonadal dwarfism.

(2) *Dwarfism and infantilism*—(a) Cachectic infantilism is produced by any chronic wasting disease, and inanition from under feeding, but severe congenital heart disease might be included in this category, (b) cachectic infantilism associated with more specific changes at the growth cartilages, such as congenital syphilis, scurvy, coliac disease, rickets, and renal dwarfism, and (c) endocrine, where arrested development results from glandular deficiency, such as is due to hypopituitary, hypothyroid and hypogonadal secretion.

<sup>1</sup> J. F. Taylor *Proc Roy Soc Med* (Sect. of Anat.), 1932, 25:111, 119.

## CHAPTER XXI

### DISEASES OF THE PANCREAS

#### DIABETES MELLITUS

**Ætiology and pathology.**—There are two possible explanations of diabetes in childhood, first, that there is a congenital deficiency of the islands of Langerhans, and secondly, that there is a damage to the existing islands by poisons or infection. *Post mortem*, even with the most careful examination by our present methods fibrosis or deficiency of the islands can only be shown in a minority of cases. In view of the fact that insulin, a product prepared from the islands of Langerhans, appears to be specific in the removal of the excess of sugar from the blood causing it to be metabolized, we must assume that diabetes is due to a dysfunction of this part of the pancreas.

Diabetes is slightly more common in males than in females. In 25 per cent of cases it is hereditary.

**Clinical picture and diagnosis.**—The disease may commence at any age but seldom below the age of one. The earliest age at which it is usually recognized is 3 or 4 years. The child is noted to be losing weight rapidly, to have an intolerable thirst and to be passing water too frequently and in excessive quantities. Emaciation may often attract attention to the condition. Other symptoms are tiredness, irritability, and hunger, but this last occurs in only one-sixth of the cases. Vulvitis is a fairly frequent initial complaint in girls. In about 15 per cent the onset of the disease appears to follow an acute infective disease, or a septic infection. Examination of the urine shows the presence of a reducing substance, which is glucose, and there may be diacetic acid and acetone. On examination of an untreated diabetic, the tongue may be red and clean, the skin dry and the palms of the hands and soles of the feet slightly yellow. A sweetish acetone smell is often present in the breath. The child is rapidly reduced to extreme emaciation.

**Blood-sugar curve.**—An examination of the blood sugar of a normal child shows that the resting blood-sugar is about 100 mgm. per cent. After a meal, or the ingestion of glucose, it rises rapidly to 180 mgm. per cent within half an hour, and within one and a half hours has fallen to normal or nearly so. The blood sugar curve of a diabetic, on the other hand, may start higher, say 150 mgm. per cent, and after the ingestion of sugar or a meal it rises steadily to 250 or 350 mgm. per cent and may even go much higher or remain there for an hour or more, settling much more slowly than the normal (Fig. 70).

From this it may be seen that the normal sugar content of the blood in diabetes is much higher than in non-diabetic children and being above the sugar threshold of the kidney, leads to a constant leaking of sugar into the urine.

**Renal glycosuria.**—Occasionally children are found who have sugar in the urine yet whose sugar tolerance curve and blood sugar turn out to be quite normal. These have a low threshold for sugar, allowing it to pass out into the urine at a lower level than normal.

**Diabetic coma.**—A child with diabetes is always liable to coma, and this may set in rapidly. The onset may be due to several causes, the commonest is an acute infection, the next most frequent are vomiting following a bilious attack, insufficient dosage of insulin, and gross departure from the prescribed diet. Yawning, drowsiness, irritability and vomiting may herald the onset. Later, air hunger is marked, and complete unconsciousness occurs. The treatment is similar to that for hypoglycæmia.

**Hypoglycæmia.**—If too large a dose of insulin is given the child becomes pale and lifeless, occasionally, he is irritable and extremely emotional. He is seized with numbness of the hands and giddiness and, if the condition is not checked, severe convulsions with unconsciousness. Other common symptoms are abdominal pain, hunger or nausea, headache, double vision, squint or actual temporary blindness. Not infrequently the attack starts abruptly with sudden unconsciousness, or a fit. When



using protamine insulin, hypoglycæmic attacks may occur in the early morning. An early warning of this is difficult in waking up at the usual time.

**Prognosis.**—Before the introduction of insulin the life of the diabetic was short. Now it appears that, with care, life can be prolonged indefinitely. The child puts on weight, develops, and appears normal mentally and in every other respect. The dose of insulin may be slightly reduced, and it is held by some observers that the pancreas shows a tendency to regenerate. In most cases, however, the activity of the islets steadily decreases, requiring a corresponding increase in insulin until about 100 units a day are needed.

**Treatment.**—The most successful treatment of diabetes is to adapt the insulin to the diet rather than adapt the diet to the insulin. Put another way, the child is given a normal diet for its age. This diet is weighed and its calorie value known so that it does not vary greatly from day to day. Insulin is given half an hour before breakfast and half an hour before supper and is gradually increased until the urine is almost sugar free. The aim should be to retain a trace of sugar in the urine. The introduction of protamine insulin, with or without zinc, has made it possible in many cases to give only one injection a day.

In treating a case of mg% diabetes a good book of food tables such, for example as that of Harrison and Lawrence<sup>1</sup> is almost a necessity.

It will be found most helpful to attempt to keep the protein low and to allow as much carbohydrate as can be controlled by a reasonable dose of insulin. The author is of the opinion that less protein is advisable than that given in Harrison's schedule (see Table XXIX) and that it is unlikely that the total of the fat suggested can be reached.

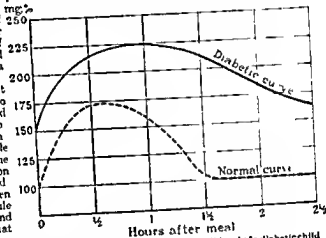


Fig. 79.—Blood sugar curve of a normal child and of a diabetic child.

TABLE XXIX<sup>1</sup>  
FOOD REQUIREMENTS OF A DIABETIC CHILD

Age in years	Ideal wt		Calories per kg body wt	Calories per diem	Cm protein per kg body wt	Daily intake in grams		
	Kg	lb				Carbo- hydrate	Protein	Fat
1	9	20	100	900	4	100	31	40
2	12	26	80	1000	3.5	120	40	45
3	14	31	80	1100	3	130	45	50
4	16	35	80	1300	3	150	50	50
5	18	40	80	1400	3	160	55	60
6	20	44	75	1600	3	180	60	70
8	24	53	75	1800	2.5	200	60	85
10	28.5	63	70	2000	2	220	60	90
12	34	75	60	2100	2	240	65	95
14	43	95	50	2100	1.5	240	65	95

<sup>1</sup> G. A. Harrison and R. D. Lawrence, Food Tables (Blumenfeld & Co.)

<sup>2</sup> G. A. Harrison, in J. Carroll Patten, Thorndike and Patten's (Discussion of Children, Arnold (1929) p. 211)

**Testing the urine**—Every specimen of urine passed—there may be six or even eight specimens per day—should be tested for diacetic acid and sugar and the insulin raised or lowered until the output of these two has been reduced to a minimum. In this way it will be found after which meals most sugar is present and then an appropriate dose of insulin can be given to control the sugar at these times on the following day. The best test for diacetic acid is the addition of iron perchloride to the urine which if diacetic acid is present assumes a dark port wine colour. The fallacy in this however is that the test is positive when alkalisites are being given and in this case the urine must be tested for acetone by the *Hofmann test* as follows. A few drops of freshly prepared sodium nitro prusside solution are added to the urine followed by ammonia until it is alkaline and then the liquid is saturated with crystals of ammonium

TABLE XXV

RECORD OF A DIABETIC CHILD FOR ONE DAY

Diabetic aged 6-8 years weight approx 47 lbs (Ht —C 160 P 71 F 87 Cal 1700)					
8 A.M.	Insulin.	If on protamine zinc test fasting specimen—the child having emptied the bladder on waking			
8.30 A.M.	Breakfast	Milk 10 oz	15	10	10
		Bread 2 oz	30	6	0
		Butter 1 oz			12.5
		Cornflakes 1 oz or	10	0	0
		Paw apple 1 oz or			
		Marmalade 1 teaspoon			
		Bacon 1 oz or egg or fish (add fat)		5	10
			50	21	37.5
11 A.M.	Lunch	Apple 4 oz. or orange 5 oz	10	0	0
12.30 p.m.	Test specimen urine (child having emptied bladder earlier)				
1 p.m.	Dinner	Potato 3 oz	15	1.5	0
		Carrot 2 oz or	2.5	0	0
		Peas 1 oz. or			
		Beet 1 oz.			
		Meat (various) 1 1/2 oz		10	8
		Pudding (Vary this carbohydrate)	10	grm car	
		eg 3 1/2 oz milk 1/2 egg	10	6.5	6
		4 oz stewed apple			
			27.5	23	14
4.30 p.m.	Insulin (if on soluble)				
5 p.m.	Tea	Milk 10 oz	15	10	10
		Bread 1 1/2 oz	22.5	4.5	0
		Butter 1/2 oz			12.5
		Apple 4 oz or pear 4 1/2 oz. or jam 1 teaspoonful	10		
		Egg 1/2 or cheese			
		Salad—tomato lettuce			
				6	5.5
			47.5	20.5	28
7 p.m. to Supper	Milk 7 oz. or orange juice, 5 oz				
7.30 p.m.	1 biscuits (plain) 2 1/2 oz (1/2 oz. each)				
			10		
8 p.m.	Test specimen if on soluble insulin				
			20	7.5	7.5
TOTAL			160	171	87

N.B.—More protein may be added if required

sulphate. If acetone is present a deep-violet colour develops in from one to fifteen minutes<sup>1</sup>. *Sugar is tested for* with Benedict's solution in the usual way. It is only by getting a specimen before each meal, i.e., before giving insulin, and testing each carefully, that hypoglycæmic attacks can be avoided and the insulin successfully regulated. As a rule, diabetics wake once in the night, and this night specimen of urine should be kept and tested. The record of a typical day in a diabetic patient's life is given (Table XXX). Whenever diacetic acid appears, the indication is to reduce the fats drastically. If much diacetic acid is present, it may be necessary to give additional sugar and insulin at the same time. The examination of the blood sugar is of practical importance both in the diagnosis and conduct of a case of diabetes. The careful examination of the urine at frequent intervals is the sheet anchor.

**Technique of administration of insulin.**—In a moderately severe or severe case of diabetes it is much easier to keep the patient free from sugar on two doses of insulin daily than on one, and the prick of the needle is soon borne extremely well by the child if the right attitude is adopted by those attending him. The inconvenience involved in giving insulin should be considered as nothing compared with the benefit which accrues from it. A different part of the body should be chosen at each administration, i.e., in the morning the arm may be used, and in the evening the buttock, all on the same side of the body. The following day this procedure may be repeated but on the opposite side of the body. The syringe and needle should be boiled at each administration, and as fine a needle as possible should be used. The skin should be sterilized with alcohol and also the top of the insulin bottle. If indurated areas appear after injections, these places should be avoided until healed.

Protamine insulin<sup>2</sup> has to some extent taken the place of ordinary insulin. The advantages claimed are that it need be given only once or at most twice per day and that it is absorbed more slowly, with consequently less tendency to hypoglycæmia.

**Management of the diabetic child.**—Certain gross errors are apt to be made in calculating any diet.

1 The composition of the various foodstuffs may obviously vary greatly.—For example, the difference in food content between a bitter and a sweet orange, a ripe and an unripe apple, old and new potatoes, fat and lean meat, is a great source of error.

2 The presence or absence of infections must be considered. A slight tonsillitis or cold causes an increased output of adrenalin, and a consequent increase in the blood sugar and depletion of the sugar stores. Extra insulin must be given to counteract this action of adrenalin. Failure to do this will result in ketosis and sometimes coma. On this account it is most important that all sources of sepsis be investigated in the diabetic child. Septic tonsils or teeth should be removed, and this can easily be done if care is taken. Four to six hours before the operation sufficient glucose to equal the caloric value of the corresponding meal (usually breakfast) is given. One to two hours before, a further dose of glucose corresponding to the next meal (lunch) is given. After the operation glucose and insulin are given again, corresponding to the normal routine. The normal diet is resumed as soon as possible.

3 The action of aperients must be taken into account. If the bowels move naturally daily a certain standard of food absorption efficiency is established. If the child becomes slightly constipated, the food is more perfectly absorbed, but on administering even a mild purge, a great wastage must take place, and attacks of hypoglycæmia threaten. Probably the best laxative is simple liquid paraffin given night and morning but if a saline is required, Seidlitz powders which contain no sugar, will be found most suitable.

4 The amount of exercise taken in the day is important. With much exercise more of the blood sugar is used up. Less insulin need then be given. Suppose a child getting 11 units of insulin before dinner daily, and having a very quiet morning is allowed to romp and play strenuously with other children for an hour preceding this meal on a particular day. This unusual occurrence will produce a marked hypoglycæmia, and, if this romp could have been anticipated, less insulin need have been given. It will be apparent, therefore, that regularity in the amount of exercise and excitement is necessary if the dose of insulin is to be the right one and attacks of hypoglycæmia are to be avoided.

<sup>1</sup> Huchison and Hunter, "Clinical Methods" 11th ed (Cassell) (1911), p. 321.

<sup>2</sup> Report of Section of Therapeutics and Pharmacology, Proc. Roy Soc Med Jan 12, 1927, xxii, 1217.

**Treatment of acute hypoglycæmia.**—Adrenalin, 6 minims, should be given hypodermically if the child is in a state of convulsion and cannot swallow. Glucose, preferably in solution, should be given rectally immediately after the adrenalin. If the child is in the early stages of an attack, lump sugar by the mouth will rapidly stop the trouble. From 4 to 8 lumps should be given and the effect noted. It is well to ascertain the cause of the hypoglycæmia attack by a scrutiny of the possible sources. Thus, further attacks can be foreseen and prevented.

## INFECTIONS AND THEIR TREATMENT

<b>Hæmolytic streptococcus</b>	In tonsillitis with cervical adenitis, otitis media mastoid disease, erysipelas impetigo pemphigus septicæmia and pneumonoma the drug of choice would be sulphamethazine sulphadiazine or sulphathiazole
<b>Pneumococcus</b>	In pneumonia particularly of the lobar type empyema otitis media meningitis and peritonitis the drug of choice would be sulphadiazine sulphamethazine or sulphapyridine
<b>Meningococcus</b>	In septicæmia and meningitis the drug of choice is sulphadiazine, sulphamethazine or sulphathiazole
<b>Gonococcus</b>	In ophthalmia neonatorum vulvovaginitis urethritis and arthritis the drug of choice is sulphadiazine sulphamethazine or sulphapyridine
<b>Staphylococcus</b>	In septicæmia and osteomyelitis, pneumonia meningitis skin infections, boils carbuncles and styes the drug of choice is sulphathiazole
<b>Bacillus coli</b>	In pyelitis and infections of the urinary tract the drug of choice is sulphamethazine sulphadiazine or sulphathiazole
<b>Dysentery bacilli</b>	In acute bacillary dysentery the drug of choice is sulphasuxidine or sulphaguanidine

In measles whooping cough scarlet fever and influenza there is no indication for the Sulpha drugs *except for the treatment of complications*

In rheumatism no good effects have been noted

In undulant fever and meningitis due to the Pfeiffer bacillus the effect is questionable

**Local chemotherapy**—The indications are the same as for adults. For wounds or burns the application of powdered Sulpha drugs is indicated but over dosage must be carefully avoided

**Prophylaxis**—This may be indicated in children before tonsillectomy and dental extractions particularly those children who have heart disease or have had scarlet fever

## METHOD OF ADMINISTRATION

**By mouth**—The drug is best given by the mouth and the method will depend on the age. To the very small infant it may be given with its feed or immediately before in a teaspoon of milk or fruit juice. For older children it may be crushed up and given in cold milk fruit juice ice cream jam honey or packed in a grape. Where children are vomiting it is still worth while giving by mouth as sufficient may be kept down to combat the infection. If not other methods must be used

**Intramuscular injection**—Where there is vomiting the drug may be given intramuscularly (but is best given intravenously) in doses similar

to those administered by the mouth. The sodium salts of sulphathiazole and sulphapyridine may be used for intramuscular injection in a 10 per cent solution. As they are irritating however, this method should be avoided.

*Intravenous administration*—Where the child is desperately ill and unable to take fluid by the mouth a rapid effect may be obtained if the drug is given intravenously. It is best diluted with saline and glucose and is often given with great advantage in an intravenous saline and glucose drip. Sodium sulphapyridine and sodium sulphathiazole are administered in a 10 per cent solution, whereas sodium sulphadiazine should be given in a fresh aqueous solution in 5 per cent strength in doses similar to those administered by the mouth.

*Intrathecal and intraperitoneal administration* are definitely contra-indicated.

*Dosage*—In theory the dose of the Sulpha drugs should be decided by an examination of the sulphonamide content of the blood. The optimum appears to be from 5 to 10 mgm per cent of the drug. Since the excretion of the various drugs varies, the level will not be equal in all. For instance sulphinguanidine and sulphasuxidine are much less readily absorbed, sulphamethazine is readily excreted and much less toxic and therefore larger doses are tolerated. It may be taken that a good working rule is

One grain of the Sulpha drug per pound body weight per day or  
One gramme per stone of body weight per day, up to the age of 10 or 12 years

*Times of administration*—The dosage should be spread evenly over the 24 hours, in order to keep the blood level at the optimum concentration. Usually, this is best accomplished by giving the drug at 4 or 6 hourly intervals day and night. For infants, it is an advantage to give the dose at feeding time which is usually 4 hourly.

*Initial dose*.—Having determined the 24 hour dose and its 4 or 6 hourly portion, it is well to give a double dose initially e.g.

A child aged 3 years weighs 30 lbs and would therefore require 30 grains (i.e. 2 grammes) of the drug spread over 24 hours. Each tablet of the Sulpha drug is 7½ grains (i.e. one half gramme) and it would be best to give him 7½ grains (½ gramme) each 6 hours day and night. The initial dose would best be 15 grains (i.e. 1 gramme).

*Length of administration*—It is not possible to lay down dogmatically a course of the drug, but it is usual to administer it for three days when the vast majority of cases of acute infection will have received the maximum benefit. It is a mistake to start with a small dose, and increase to a maximum dose; the reverse should be the practice. In chronic infections, such as sub-acute pyelitis, long standing ear discharge, ulcerative colitis and other bowel infections it is often best to give the appropriate drug in a short course of three days each week or fortnight. In this way the organisms do not appear to become resistant, as they do when the drug is given over prolonged periods, without a break.

*Excretion*—This varies both with the individual and the solubility of the particular preparation. For instance, sulphamethazine is highly soluble and readily excreted. Much fluid should be given by mouth while

any of the Sulpha preparations is being administered. The object is to keep the drug in solution and prevent damage of the kidney by formation of crystals in it. It is said that the administration of alkalis facilitates this. Certainly, two pints or more of fluid should be aimed at in each 24 hours.

The Sulpha drugs are excreted in the saliva, in the sweat and also in the milk of nursing mothers, and they pass through the placenta into the foetal circulation. These facts should be borne in mind when administering the drug to a pregnant or lactating woman. Apparently, the drug is well tolerated by both the foetus and suckling infant.

### TOXIC REACTIONS

General malaise, with loss of appetite, drowsiness, and occasionally even mental confusion sometimes occurs. These symptoms are seldom encountered with sulphadiazine or sulphamethazine.

Vomiting is probably the commonest complication. In some cases it appears to be due to a local gastritis, but it is usually a toxic effect, and in children it is rarely found when sulphadiazine or sulphamethazine are used. It is claimed that alkalis, such as bicarbonate of soda or citrate of soda or meotinamide in doses of 10 or 15 grains, lessen the vomiting if administered with the Sulpha drug. This has not been the author's experience. Where the vomiting is severe it is well to miss the next dose, but if this manoeuvre fails it may be necessary to give the drug intramuscularly or intravenously.

Rashes may be erythematous or morbilliform (see Fig 57, p 299). There is often a temperature and the condition may be mistaken for measles or scarlet fever. No Koplik spots are to be seen, however, and the glands are not enlarged. When a rash occurs it is best to miss out or reduce the next dose, but in order to prevent sensitisation, the drug should not be entirely discontinued. It is generally considered that children receiving one of the Sulpha drugs should not be unduly exposed to strong sunlight or to X rays, as they may be photo-sensitive.

Cyanosis may occur, with the formation of methæmoglobin or sulphæmoglobin. It should not be looked upon as a serious symptom. It is not, apparently, due to eating eggs and other sulphur containing foods, as was formerly thought.

Drug fever may occasionally appear after the temperature caused by the original infection has subsided. This seldom occurs before the eighth day, if the drug is withdrawn the temperature subsides at once.

Leucopenia and anaemia very occasionally develop. Usually, there is little or no reduction of the white or red cells in children. As a general rule, a three- or four-day course of one of the Sulpha drugs should be looked upon as perfectly safe. The author has never seen hæmolytic anaemia in a child as the result of chemotherapy, although it has been recorded.

Anuria and hæmaturia appear to be due to a mechanical blockage of either the tubules or the ureters by bundles of crystals. The more insoluble sulphic nanudes are responsible for most cases. Anuria is extremely uncommon in children, cystoscopy with mechanical clearing of the blocked ureters may be found necessary. Hæmaturia is found in perhaps

**Example** A ten pound infant would require 10,000 units in the 24 hours and would therefore get 1250 units each three hours, day and night, for three or four days or more

Provided adequate access to all infected parts is possible then local administration is a success. It can be administered as the sodium or calcium salt of penicillin or along with sulphamide powder locally. It can be administered locally as a powder, a spray or a wet dressing.

**Results of treatment.**<sup>1</sup>—Florey states that out of over 300 cases of chronic sinusitis, abscesses, arthritis, skin infections, infections of the eyes and lips, empyemata, acute mastitis and infections of the hands, particularly staphylococcal infections, 250 had shown complete recovery, 41 had improved and in 14 no change had been registered. It fails to cure subacute bacterial endocarditis. Empyemata and meningitis are treated by injections into the pleural cavity and spinal canal. Pulmonary actinomycosis is also treated. Colbrook and his colleagues have found penicillin cream highly effective in eliminating infections from burns.

Discussed at the Royal Society of Medicine Nov. 3, 1943 on penicillin *Brit. Med. Jour.* Nov. 20, 1943, II, 734.

Lawes (Editorial), October 31, 1943, II, 610.

A. Fleming *Brit. Jour. Exp. Path.* 1943, 24, 6.

A. Fleming, *In vitro* Tests of Penicillin Potency *Lancet* June 20, 1943, I, 73.

Report on the Therapeutic Properties of Penicillin *Brit. Med. Jour.*, April 15th, 1944, I, 813.



**Treatment**—The mother should be reassured and told to put drops of liquid paraffin or some salt vaseline up the nose on the affected side for a few days. The duct opening will then become less milium and patency will be re-established. Usually nothing further is required but meanwhile the eye may be bathed twice daily and a little golden ointment (yellow oxide of mercury) may be applied at bedtime.

### INFLAMMATION OF THE CONJUNCTIVA

In its mildest form, namely *congestion* or *hyperæmia*, this may be due to (a) foreign bodies (b) glare of strong light (c) irritants such as smoke or gaseous products in the surrounding atmosphere (d) illness as in hay fever (e) blocking of the lacrimal duct.

**General treatment**.—This consists in removing the cause.

**Local treatment**.—This consists in bathing the eyes with boric lotion or zinc sulphate solution, one grain to the ounce. Sedatives such as cocaine solution or adrenaline give only temporary relief.

### ACUTE CONJUNCTIVITIS

(COLD IN THE EYE    PINK EYE)

In acute conjunctivitis the discharge is muco-purulent. The eye is fiery red and congested and the vessels engorged. The lids are stuck together in the morning and the lashes are matted.

**Progress**.—Usually such infections reach their height in three or four days and are better in a week to ten days.

**Ætiology and pathology**.—The organism most commonly found is the 'Koch Weeks bacillus', which is a slender Gram negative rod. It is infectious to others but is killed on drying. Diplococci indistinguishable from pneumococci or pneumococci themselves also cause epidemics of conjunctivitis. The bacillus of Leffler may be found in some epidemics. Acute conjunctivitis may be caused by the *Staphylococcus aureus*. Conjunctivitis is a common complication of measles and scarlet fever.

**Treatment**.—Frequent bathing with 1:10,000 perchloride of mercury should be undertaken at once. In infants and young children the lids should be held apart and the lotion dropped in freely. Boric ointment or vaseline is smeared along the lids at bedtime and the eyes should not be bandaged. Protargol 20 per cent. or Argyrol 25 per cent. may be used if the condition is not clearing up rapidly enough. One of the sulphonamides such as Albucid drops will be found useful.

### OPHTHALMIA NEONATORUM

This is a preventable disease and responsible for 50 per cent. of blind children and 7 per cent. of all blind persons (Parsons)<sup>1</sup>. The infant is infected during birth from the mother's passages or discharges. The eye infection is noted about the third day, both eyes being involved as a rule. The lids are swollen and there is much congestion. There is a risk of corneal ulceration and even perforation.

<sup>1</sup> Sir J. H. Parsons, "Diseases of the Eye," 30 Ed. (Churchill), 1938.

A. Borsby *et al.* Fluoropyridine in *Ophthalmia Neonatorum*. *Brit. Med. Jour.* March 7 1941, 322.

in 510 cases showed evidence of tuberculous infection in the chest in 72.2 per cent nearly 9 times that of a series of controls. Contact evidence was obtained in 29.9 per cent of the cases and blood sedimentation rate was raised in 79.8 per cent. Following these cases up for 10 years 5.3 per cent developed clinical tuberculosis and 0.8 per cent died. Phlyctenular conjunctivitis appears to be a manifestation of tuberculous infection due to the development of an allergic reaction to this particular organism.

**Clinical picture.**—Along with the congested conjunctiva there is a tendency to blepharospasm so that the eyes are sometimes kept tightly closed for days on end. This spasm may be present even in a dark room and is only relieved by the application of cocaine ointment or drops. Actual photophobia may also be present.

**Recurrence.**—There is a marked tendency to recur at intervals particularly in the spring.

**Treatment.**—Local treatment by bathing the eye in boric lotion, and the application of yellow oxide of mercury appear to be all that is necessary.

**General treatment.**—Attention should be given to infected teeth and tonsils and the supply of a generous well balanced diet. All the vitamins should be given but particularly those found in cod liver oil. Fresh air and iron tonics with frequent changes to the seaside are indicated. No relaxation in the supply of cod or halibut liver oil and the maintenance of a high standard of health should be allowed otherwise recurrence will invariably take place.

### ANGULAR CONJUNCTIVITIS

The area affected is limited to the intermarginal strip of conjunctiva at the inner and outer canthus.

**Ætiology.**—The organism is the bacillus of Morax Axenfeld, a rod shaped Gram negative diplobacillus.

**Treatment.**—7mc sulphate 2 grains to the ounce is specific.

### INFLAMMATION OF THE CORNEA

(KERATITIS)

This may be due to a variety of organisms and causes acute ulceration. The advice of an eye surgeon should be sought at once, as the condition is serious since perforation or permanent clouding of the cornea with partial blindness may follow.

#### PHLYCTENULAR KERATITIS (STRUMOUS KERATITIS)

This condition is merely an extension of phlyctenular conjunctivitis to the cornea where it may give rise to ulceration and permanent opacities or nebulæ with varying degrees of blindness.

**Treatment.**—This is as for phlyctenular conjunctivitis.

**Interstitial keratitis (syphilitic keratitis).**—This has been described on p. 349.

### DISORDERS OF THE EYELIDS

#### BLEPHARITIS

This may be acute accompanying an acute or chronic conjunctivitis. The chronic form is often found in eczematous watery or strumous children.

*Source: Hanburry and Denham "Phlyctenular Keratitis," Brit Med Jour April, 1936 1: 80"*

## CHAPTER XXIV

### INTESTINAL PARASITES

Four intestinal parasites are commonly found in infancy and childhood in the British Isles. These are

- (1) *Tænia mediocanellata* (tape worm)
- (2) *Ascaris lumbricoides* (roundworm)
- (3) *Oxyuris vermicularis* (threadworm) and
- (4) *Giardia (Lambia) intestinalis*

#### TÆNIA MEOIOCANELLATA (TAPEWORM)

This is a relatively common intestinal parasite but the source of infection is very often difficult to ascertain. Occasionally a dog or cat has been found to be the infecting agent and in some cases the patients or their parents will be found to have recently returned from the East. It is commonest between the ages of 2 and 8 years. The worm varies in length from 5 to 9 yards and is segmented, the segments being yellowish white, flat and in the centre of the worm broader than they are long whereas towards the head and tail they are longer than they are broad. As the head is approached the segments rapidly become elongated and very slim and in the last few inches are rarely more than  $\frac{1}{8}$  inch across, although they are about  $\frac{1}{4}$  inch long. The head itself is no larger than the head of a pin and the neck leading from it is about the thickness of a pin. Thus finding the head and neck though a matter of great importance is also one of extreme difficulty. The worm fastens itself with its head beneath the folds of the bowel high up. Its site probably varies however though in the most difficult cases it is usually no great distance from the duodenum. The lower down the bowel it is the more easily it can be dealt with.

**Symptoms and diagnosis.**—It may be noticed that the child is wasting or is suffering from bouts of colic, passing large, mucus-containing stools. Some mothers claim that the colour of their children is yellowish or earthy, certainly they are not as good a colour as usual. Then, segments may be noted in the stools either singly or two or three fastened together each probably the size of an average finger nail and somewhat similar in shape. This makes the diagnosis simple. Segments are passed in almost every motion and occasionally are found in the bed.

**Treatment.**—Once the diagnosis has been made, treatment should be commenced. It is important to follow the details rigidly, the following treatment being suitable for a child aged 5 to 7 years.

**The first day.**—The diet is light and one which leaves as little residue as possible. For breakfast, groats, porridge or other cereal accompanied by some sweet preparation such as syrup or honey, is suitable and weak tea or skimmed milk should follow. At midday the child should have soup

A fully developed worm is 6 to 8 inches in length and, except for the fact that they are much larger, they resemble the ordinary earthworm.

Children infected with ascaris appear in moderately good health. They are restless and show loss of appetite and undigested and mucus-containing stools but, on the whole, are comparatively well nourished notwithstanding. Worms are not present in large numbers, an occasional one or two being passed at infrequent intervals. At times they are ejected from the mouth. This migration is dangerous because of the possibility of respiratory inhalation. The worms may be found also in the bed or may be passed by themselves without any motion.

**Treatment**—The diet should be similar to that described for tapeworm, namely low in protein and fat, and high in carbohydrate, so as to have very little residue.

A mixture containing santonin 11 grains, compound scammony powder 2 grains, calomel  $\frac{1}{2}$  grain should be given at bed time for three nights and first thing in the morning a tablespoonful of milk of magnesia or a teaspoonful of Frob's Fruit Salts should be administered.

Care should be taken that no oily substance such as castor oil or olive oil is given during this period, otherwise santonin poisoning, with coloured vision and vomiting is likely to occur.

### OXYURIS VERMICULARIS

(THREADWORM)

The threadworm is white,  $\frac{1}{4}$  to  $\frac{1}{2}$  inch long and about the thickness of a fine cotton thread.

**Incidence**—This is said to be 43 per cent (Franklin 1942, Cram, 1943) of the general population.

**Sex**—They are more commonly found in girls than boys.

**The worm**—Cram (1943) claims that a single worm may turn out an average of 11 000 eggs. The eggs are found in the dust of rooms and clinging to the walls. As a rule not less than one other individual in each family is infested and usually all members are infested. The diagnosis is made by finding the threadworms or the eggs either in the stool or from the perianal region which has been swabbed with a cellophane-tipped swab. The cellophane is detached from the rod mounted in water or 1% normal sodium hydroxide solution and examined under the microscope for the eggs. The swab is best taken immediately after the patient gets up in the morning, and before bath or motion. It is generally considered that seven negative perianal swabs are necessary before the child is considered cured.

**Symptoms**—The chief symptom is irritation about the anus and vulva and threadworms may be considered a common cause of vulvo-vaginitis. Other symptoms are restlessness and sleeplessness from the irritation, vague abdominal pain and a tendency to unexplained diarrhoea. Thumb-sucking and nail biting tend to cause reinfection. Nose picking and habit spasm are not symptoms of infection with threadworms. On examination

on the following dosage of atebum quinacrine, i.e. mepacrine hydrochloride B.P. given orally

<i>Age in years</i>	<i>Daily dose in 0.1 gramme tablets</i>	<i>Course in days</i>	<i>Total dose</i>
$\frac{1}{2}$ -2	$\frac{1}{2} \times 2$	3-5	0.15-0.25 grammes
2-6	$\frac{1}{2} \times 2$	3-5	0.30-0.50 "
6-9	$\frac{1}{2} \times 3$	4-5	0.60-0.75 "
9-10	$1 \times 2$	5	1.0 grammes
Adult	$1 \times 3$	5	1.5 "

The cysts disappear from the stools in 5 days as a rule and the diarrhoea clears up. The drug is non-toxic but the child may very occasionally become yellow and if he should vomit the stain is permanent on the bed clothes. A further course is sometimes necessary.

## APPENDIX II

### MILKS, PROPRIETARY FOODS &c

COMPOSITION OF THE BETTER KNOWN CONDENSED (EVAPORATED) MILKS

Name of Milk		Calorie value of 1 ounce	Fat per cent	Protein per cent	Carbo- hydrate per cent	Water per cent
<b>A SWEETENED FULL CREAM CONDENSED MILKS</b>						
Nestlé's		102	10.2	9.4	53.0	25.3
Diploma		101	9.6	8.4	54.62	25.5
Berna		103	10.5	9.5	53.0	20.0
Tip-top or Red Butterfly		98	9.1	8.54	52.86	20.9
<b>B UNSWEETENED FULL CREAM EVAPORATED MILKS</b>						
Ideal		51	9.5	8.84	12.21	17.5
Carnation		48	9.2	8.6	11.1	69.4
Libby's		50	9.2	9.6	11.5	18.0
Begal or Coronet		50	9.6	8.32	12.02	18.2
Berna		49	9.5	9.5	10.0	69.0
Everyday or Green Butterfly		50	9.1	8.85	12.20	17.9
Pet Milk		42	8.0	7.6	9.7	73.4
<b>C SWEETENED SKIMMED CON- DENSED MILKS</b>						
Moonraker		84	0.55	9.76	59.84	27.6
Blue Butterfly		84	0.4	10.0	58.8	28.8

- Publum*—(P), 14.8, (F) 3.00, (C), 71.4—Composed of wheatmeal, cornmeal, rolled oats, wheat germ, alfalfa, yeast, and edible bonemeal. Calorie value of 1 oz. = 120.
- Ridge's Food*—(P), 12.17, (F), 2.71, (C) 79.72—A baked flour, containing only 1 per cent of soluble carbohydrates, the remainder being starch. Recommended to be made with milk and water. Calorie value of 1 oz. = 116.
- Robinson's Patent Barley*—(P) 7.2, (F), 1.35, (C), 81.3—Ground pearl barley, poor in every element except starch and mineral matter. Calorie value of 1 oz. = 109.
- Robinson's Patent Groats*—(P), 12.25, (F), 7.3, (C) 72.4—Ground oats from which the husk has been removed. Rich in protein and mineral matter. Calorie value of 1 oz. = 121.
- Ryala Crispbread*—(P) 11.6, (F), 1.3, (C), 74.8—Made in England from crushed whole rye grain. Calorie value of 1 oz. = 104.
- Savory & Moore's Food*—(P) 12.6, (F) 1.5, (C), 76.8—Composed of wheat flour with the addition of malt and diastase. When prepared according to the directions most, but not all of the starch is converted into soluble forms (chiefly maltose and malto-dextrins). One or two tablespoonsful (equals from 1 to 2 oz.) to be mixed with two or three tablespoonsful of cold milk or milk and water, and  $\frac{1}{2}$  pint of boiling milk or milk and water to be added. Calorie value of 1 oz. = 111.
- Sister Laura's Food*—(P) 20.06, (F) 2.94, (C), 75.27—A food prepared from wheat starch, intended to be added to undiluted milk. Calorie value of 1 oz. = 123.
- Soya Bean Flour*—(P) 4.4, (F) 20, (C) 14—A flour with a very high food value, especially protein, and a high vitamin content, made from the soya bean. Calorie value of 1 oz. = 121.
- Vegeta*—(P) 9.63, (F) 2.90, (C), 77.24—A dried mixture of spinach, carrots and tomatoes, rich in vitamins A, B and C (the latter not destroyed by drying). The vehicle used is a mixture of starch and its various cleavage products obtained by diastatic disintegration. To be used as a substitute for home made vegetable broth. Calorie value of 1 oz. = 112.
- Vital*—(P), 7.50, (F) 11.60, (C), 56.33—Composed of marrow fat, glycerin, extract of red bone marrow, eggs, salts of lime, etc., malt extract, and the juice of fresh lemon.
- Wheat Wheat*—(P) 11.52, (F) 7.66, (C) 74.77—Made in England from whole wheat. Calorie value of 1 oz. = 127.

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